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Original Articles.

THE BI-POLAR CELLS OF THE SPINAL CORD AND THEIR CONNECTIONS.

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EXAMINATION of the spinal cord of man in reference to the connections of the cells of Clarke's column, the intermedio-lateral, and the solitary cells of the posterior horn, is attended with many difficulties, which the anterior cornual cells do not offer. The latter, multipolar cells, give origin to the large fibres of the anterior roots, and are connected with similar cells above and below, and in the opposite anterior cornu. It would seem probable that their multipolar character is associated with these connections. The other cells of the cord are, for the most part, simply bi-polar. There is good reason to believe that nerve fibres bear a relation in size to the cells in which they originate. It is also probable that a nerve cell is larger when the nerve formed by its axis-cylinder process has a long course before it comes to its final termination. Hence the anterior cornual cells of the lumbar or cervical enlargements are larger than are met with elsewhere. A fibre, however, may branch after leaving the cell, as happens in the T-shaped fibre of the cells of the ganglion on the posterior root (Ranvier). This, however, does not apply to the fibres met with in the anterior roots, and it may be concluded that Gaskell[1] is

[1] 'Journal of Physiology,' vol. vii.

right in regarding small fibres as originating from small cells. He has shown that in the anterior roots throughout the dorsal region are numerous *fine medullated fibres* (leucenteric). These also are found in the second sacral root, and in the upper cervical region, and form the viscero-vascular rami communicantes. If his first statement be accepted, that small fibres arise from small cells, the second statement that they originate from Clarke's column, which undoubtedly agrees in its distribution with this outflow of fine fibres by the anterior roots, cannot be accepted, because the cells of Clarke's vesicular column are by no means small, measuring where the column is well developed, 90 μ, and even the smallest cells (40 μ) are larger than the cells of two other groups, which he admitted might possibly be the source of fine fibres, and which I shall endeavour to show is much more probable. In fact, although text books state that Gaskell[1] has proved that these fine efferent fibres originate in Clarke's column, the whole evidence to support this hypothesis rests upon the assumption that because the cells of this posterior vesicular column are so placed in the cord as to agree in situation with the outflow of these fibres, therefore they give origin to them. No microscopical evidence tends to support this view. Occasionally fibres may be seen running from Clarke's column to the anterior cornua; especially have I noticed this in the mid-dorsal region. But the fibres cannot be traced into the anterior roots, and it may be that some of them pass into the anterior commissure as Bechterew states. Even this does not show that the fibres have originated from the cells of Clarke's column—in fact, they may be merely fibres

[1] Dr. Gaskell sums up his views of the functions of the various groups of the spinal cord:—(*a*) Cells of anterior horn = nucleus of efferent nerves to somatic muscles. (*b*) Large cells of lateral horn = nucleus of efferent nerves to striated splanchnic muscles. (*c*) Cells of Clarke's column = nucleus of anabolic (inhibitory) nerves to splanchnic glandular system and to muscles of viscera and vascular system. (*d*) Solitary cells of posterior horn = nucleus of motor nerves to muscles of viscera. (*e*) Small cells of lateral horn = nucleus of katabolic (motor) nerves to splanchnic glandular system and to muscles of vascular system.—"On the Relation between the Structure, Function, Distribution and Origin of the Cranial Nerves; together with a Theory of the Origin of the Nervous System of Vertebrata." By W. H. Gaskell, M.D., F.R.S. 'Journal of Physiology,' vol. x.

passing out of the postero-external column on their way to the anterior horns.

Microscopical examination, however, shows beyond a doubt that fibres of the postero-external column, coming from the sacral and lumbar nerves, have their station in the lower part of Clarke's column. Physiological experiments in monkeys and pathological investigations in cases of injury and diseases of the roots of the cauda equina show degeneration of the fine nerve plexus around the cells of the posterior vesicular column.

The evidence of the lower connection of this group of cells will be first considered microscopically. If the cord of a fœtus be examined in the lower dorsal region, sections stained by Weigert's method show bundles of fibres passing out of the postero-external column into the posterior horn like a brush (*vide* micro-photograph 1.), and limiting on the outside a group of cells lying in neuroglia substance. The cells are not larger than the adjacent solitary cells, and there is very little evidence of any fine plexus of nerve fibrillæ around them. The cells measure at this period about 20 μ (average).

At successive periods of growth of the spinal cord, namely, four months, nine months, two years, and adult, transverse vertical sections showed the cells to be slowly increasing in size. At two years they are three times as large, 60 μ, and in the adult even more than that, measuring on an average 70 μ (*vide* micro-photograph 2). The latter measurement agrees pretty accurately with the average size determined by Sherrington.[1]

In the spinal cord of a child two years old I was fortunate enough to obtain a section showing fibres passing out of the postero-external column, breaking up into a fine brush of fibrillæ, with varicosities thereon around the cells of the column. (See fig. 2.)

Examinations of other sections from a very well-hardened adult spinal cord, made in a vertical direction, showed the

[1] Philosophical Trans. R. Society, vol. clxxxi., p. 33-48; 'Outlying Nerve Cells in the Mammalian Spinal Cord,' by C. S. Sherrington, M.A., M.B.

cells placed with their long axis vertical, lying in lymph spaces, surrounded by a spongy network of fine nerve fibrillæ. These could be traced across the lymph space into actual protoplasmic continuity with the cell. (See micro-photograph 3.)

It is possible that the cells stain a sepia colour by Weigert's[1] method, on account of the cell being covered with these delicate fibrils, the optical section of which give it its fine granular appearance. This fine plexus has long been known to be atrophied in cases of tabes dorsalis, and in injury of the roots of the cauda equina, associated with a sclerosis of the fibres of the postero-external column. The striking features of tabes which may be associated with this pathological change, are absence of the knee jerks, and loss of the guiding sensations, which normally operate in a reflex manner in the production of unconscious equilibration. Visceral disturbances are not at all uncommon, and Dr. Ross was the first to suggest that visceral crises and other disturbances were due to affection of the cells of Clarke's column.

It is possible, as suggested by Dr. Ferrier,[2] that along with tactile, common sensory, visual or labyrinthine impressions, other sensory impressions are correlated in the cerebellum, with the motor adjustments necessary for stability and equilibration. It may be that visceral or organic sensory impressions are represented in the cerebellum, but it is quite probable that other smaller cells in the immediate neighbourhood of Clarke's column, namely, the solitary cells, or the cells of the intermedio-lateral tract convey these organic sensations. Lissauer and Krauss, Oppenheim and Sœmmerling,[3] and others, have shown that the earliest change in locomotor ataxy is an atrophy of this fine nerve plexus. I described a case of injury to the roots of the cauda equina which showed this condition, together with a sclerosis of the fibres entering it from the

[1] Ct. Lenbossek, *Archiv. Microsk. Annat*, v. xxxiii., 1889.

[2] *Functions of the Brain.*

[3] Krauss, *Zur Pathol. Anat. der Tabes Dorsalis, Neurol. Centralblatt*, Feb. 1st, 1885; Lissauer, *Archiv. fur Psychiatrie u. Nervenkrankheiten*, 1885; Oppenheim and Sœmmerling, *Archiv. fur Psychiatrie* vol. xx.

postero-external column.[1] The cells were somewhat shrunken, but there was no degeneration in the direct cerebellar tract. Cases of tabes have been mentioned to me in which the cells of Clarke's column were absent, but there was no degeneration in the cerebellar tract. This was due, as afterwards found at my suggestion, to the cells having tumbled out owing to the atrophy of the supporting fine plexus. For when the same cord was examined, after imbedding in celloidin, the cells were found to be as numerous as in normal sections.

With a view of determining the connection of the cells of Clarke's column with the fibres of the posterior roots, I made the following experiments:[2]—Unilateral section of the posterior roots of the cauda equina was made in two monkeys, the lower lumbar and sacral nerves being divided. The animals lost sensation in the lower limb of the same side, and there was absence of the knee-jerk. They were killed at the end of a month with chloroform. On microscopical examination of sections stained by Weigert's method the following degenerations are observed:—In the lower lumbar and upper sacral segments corresponding to the roots divided, there is complete degeneration of all the fibres entering the posterior horn, and a uni-lateral degeneration of the whole posterior column, except a small triangular area at the outer extremity of the posterior median fissure. Neither internal, central, nor external root fibres are seen, and there is a complete absence of the border zone of Lissauer. Moreover, an ascending bundle of small fibres seen at the outer part of the posterior horn of the uninjured side is quite absent. At the junction of the first and second lumbar segments the root fibres are seen undegenerated, and a triangular wedge of undegenerated fibres makes its appearance to the median side of the posterior horn. An examination of Clarke's column at this level shows an absence of the fibres which

[1] 'Journal of Anat. and Physiology,' vol. xxii., "On the Microscopical Examination of Clarke's Column in Man, the Monkey, and the Dog."

[2] These experiments were performed at the Brown Institution in the year 1888, by the kind permission of Professor Horsley.

pass from the postero-external column, and on the side of the lesion atrophy of the fine fibrillary plexus in which these fibres terminate. The number of cells is about equal on the two sides, but the difference in appearance of the fine fibrillary plexus was most marked in every section, notwithstanding the great number examined. The fibres which enter Clarke's column from the postero-external column may be divided into two sets, those which come from the outer division of the inner root fibres, and those which seem rather to limit Clarke's column than enter into the formation of the fine fibrillary network. Just above the lesion these fibres which run on the outside of the column, begin to appear, but still the fine nerve network around the cells is atrophied for some considerable distance above the lesion. In fact, it is not until we come to the seventh dorsal segment, where Clarke's column has become comparatively small, that the fine fibrillary plexus appears equal on the two sides. Coincident with this is an absence of all degeneration except in Goll's column; that is to say, not until the degenerated fibres have all disappeared from the postero-external column, does Clarke's column, as regards its fine plexus of fibrillæ appear the same on the two sides. The degeneration of Goll's column can be traced up to the post-pyramidal nucleus. The area of degeneration is smaller the higher it exists in the cord—probably due to the fibres themselves becoming smaller.

The degenerations are indicated in figs. 1 and 5. It seems probable to me that most of these large fibres which end in Clarke's column, and which degenerate when the lower roots of the cauda equina are divided, are supplied by the nerves of the lower extremity. These results accord with those of Tooth, with regard to the area of degeneration resulting from unilateral section of the roots of the cauda equina.[1]

Lissauer and Krauss, from their observations on the degeneration of the spinal cord in tabes, conclude that those fibres which lie most internally are most degenerated; and it is remarkable that the fibres which are most degenerated

[1] "Degenerations of the Spinal Cord," Gulstonian Lectures, 1890, Howard Tooth.

come from the lower roots, while those which form the outer group come from roots higher up.

Sherrington,[1] in an exhaustive account of the outlying nerve cells of the mammalian spinal cord, makes the following statement :—

"That the cells of Clarke's column are connected on their proximal side with the fibres of the cerebellar tract admits of little doubt; as to their distal connections it may be said that two views are current. The one which has been supported by Mott is that the cells are connected with afferent fibres—fibres of the posterior root. The other (Gaskell, Hill) is that Clarke's column is connected with the efferent fibres of anterior roots. Of these views the former, I think, derives support from the facts observed with regard to outlying cells in the posterior root zone of the cord. But it is difficult to believe that these outlying cells, although they appear equivalent to members of Clarke's column, can be at all closely connected with any fibres in the anterior root. Indeed it appears more likely that not only the vesicular group of Clarke, and the outlying cells of the external posterior column, but also the cells appearing in or near the gelatinosa, all belong to the afferent system entering by the posterior root."

Histology, physiological experiment and pathological observations all tend to prove that the cells of Clarke's column are brought into relation with fibres of the postero-external column by means of the fine nerve network in which these fibres end.

We have now to consider the upper connection of Clarke's column. As already stated, histological observations tend to show that the cells of Clarke's column give off large fibres, which run upwards, slightly forward, and then outwards through the lateral column, to reach the direct cerebellar tract of the same side. Pathological observations strongly support this view, although Tooth[1] places the point of origin of the fibres in the direct cerebellar tract as somewhere between the ninth and eleventh dorsal, and states that it is probably higher in the monkey. He also concludes that the posterior vesicular column is in connection with fine fibres, because compression of the cord below the ninth dorsal (where the column is best

[1] *Loc. cit.*

developed) has, in a number of instances, not produced any degeneration in the direct cerebellar tract. He argues, therefore, that these cells cannot be the source of the fibres of this tract, but rather are connected with fine fibres which may ascend in the posterior median and antero-lateral. With regard to the former conclusion, neither pathology nor experiment supports it. I have lately recorded a case in which there is complete degeneration of the whole of Goll's column, with no degeneration elsewhere in the cord, Clarke's column being perfectly normal. Neither does his conclusion agree with the results of unilateral section of the posterior roots.[1]

Finally, Dr. Tooth (page 60) states that the "*true* direct cerebellar tract consists of large fibres, the antero-lateral principally of small ones. The direct cerebellar tract derives its fibres from the posterior roots, the antero-lateral from the grey matter of the cord—at any rate not from the posterior roots." I must confess that I cannot understand, if this be so, why no degeneration of the direct cerebellar tract results from section of the posterior roots of the cauda equina. The reason why degeneration of the direct cerebellar tract does not occur unless the compression is at the ninth dorsal or above it may probably be accounted for in this way:—the tract itself does not exist as a collection of fibres on the periphery of the cord until we reach the ninth dorsal, because the fibres which form it have to pass upwards, forwards, and outwards through the lateral column. A transverse section[2] of Clarke's column shows numbers of large fibres cut transversely. Moreover, the cells are placed, except in the lowest part, with the long axis vertical. These large fibres I believe to be the medullated axis cylinder processes of the large bi-polar cells. The very position of the posterior vesicular column in the centre of the cord would necessitate a severe crush in order to destroy the cells and their fibres. Whereas a much slighter compression, for example by a tumour,

[1] *International Journal of Medical Science*, January, 1891.

[2] *Vide* micro-photograph 2.

higher up, where these fibres have come to the surface, would more readily injure them. The evidence which supports the view that the cells of Clarke's column give off fibres which are continued upwards as the direct cerebellar tract is supported by the study of the development of this tract in the fœtal spinal cord by Flechsig, Bechterew, and others. I have myself observed in the fœtal spinal cord, passing out across the base of the posterior cornu, numbers of fibres which can be traced outwards through the front of the still unformed pyramidal tract. Vertical and transverse sections of the spinal cord in the lower dorsal region show large fibres passing out from the large bi-polar cells of this column, but they cannot be traced throughout the whole extent of their course, because they run upwards, forwards and outwards, and therefore, whether the sections be made transverse or vertical, their continuity must be severed. The fibres cannot, therefore, be traced directly from the cell into the direct cerebellar tract. With the appearance of the cells of Clarke's column in the lower dorsal region I have noticed large fibres scattered through the lateral column. To make more certain of this connection I made the following experiment. Unilateral section of the posterior-vesicular column was effected in a monkey at the level of the twelfth dorsal and first lumbar segments, and the animal was killed with chloroform after twenty days. A microscopical examination of the cord after hardening in bichromate of ammonia and staining by Weigert's method showed that the posterior column on the side of the lesion was almost completely destroyed; at the same time there was destruction of the grey matter on that side. (See micro-photograph 4.)

Microscopical examination of the sections with a high power showed, on the same side as the lesion, a complete atrophy of Clarke's column and disappearance of the nerve cells and fibrillary plexus at the level of the twelfth dorsal and first lumbar segments (*vide* micro-photo 5). At the ninth and tenth dorsal degenerated fibres were seen coursing out through the lateral column, and above this they were seen to reach the periphery; at the sixth dorsal the whole of the

degenerated fibres *had* reached the position of the direct cerebellar tract (*vide* micro-photo 6), and there were now no degenerated fibres seen in the cross pyramidal, thus coinciding with the experiment of Dr. Sherrington[1] upon descending degenerations. He found that a number of large fibres in the cross pyramidal tract of the lower dorsal region did not undergo degeneration; in all probability they were these fibres. Another fact which supports Dr. Sherrington's views is that the degenerated fibres in the direct cerebellar tract were all very large, but there were a number of smaller fibres mixed with them undegenerated, probably belonging to the cross pyramidal system. The degenerated fibres were traced up into the cervical region. In these were some few degenerated fibres on the other side of the cord, probably due to the lesion not being absolutely unilateral.

Two other experiments were made.[2] In one the unilateral section was made at the eighth dorsal. This produced only slight degeneration of the direct cerebellar tract, but there was considerable scattered degeneration in the antero-lateral. In another, unilateral injury of the postero-external column was produced about the level of the first and second lumbar, but the grey matter was not injured; in this case there was no degeneration of the direct cerebellar tract. I did not observe any visceral vascular disturbances in these animals. Of course the great difficulties attending such an experiment as the incision of Clarke's column are almost insuperable on account of the effects produced by injury of other portions of the cord; but the principal portion of the cord injured is the postero-external column, which has to be cut through in order to reach the vesicular column of Clarke. Section of the roots, however, has been shown to cause complete degeneration of the posterior column without any change in the direct cerebellar tract. It may therefore be reasonably concluded that *the cause of the degeneration of the fibres of the direct cerebellar tract is the destruction of the cells of Clarke's column.*

[1] 'Journal of Physiology,' vol. x., No. 5.

[2] The result of these experiments were made the subject of a communication to the Physiological Society, November, 1889. At the same time the microscopical specimens were exhibited.

Clarke's column extends from the first dorsal to about the second lumbar. It is best developed however in the lower dorsal region. A few somewhat similar cells are found in the sacral region, termed "Stilling's nucleus," and a few in the upper cervical region. Drs. Gaskell and Hill believe that the homologue of Clarke's column in the medulla is the nucleus of the spinal accessory and vagus.

The cells of these "nuclei" are spindle-shaped bi-polar cells—not unlike the cells of Clarke's column, only not so large. And as we know that the lateral horn contains similar cells, which moreover correspond in size, I should be inclined to believe that the larger bi-polar cells seen in vertical and transverse sections of the lateral horn in the dorsal region of the cord corresponded more closely with the cells of the vagus accessory nucleus. The homologue of Clarke's column in the medulla is probably the "cuneate nucleus," the cells of which resemble in shape and size the average-sized cells of Clarke's column. Upon this point Sherrington[1] and I concur.

Dr. Alexander Bruce[2] showed sections of the medulla of the fœtus, which demonstrated that the upward continuation of the "cuneate nucleus" was "Deiter's nucleus," and the cells of this nucleus are in connection with the fibres coming from the semi-circular (deep) root of the auditory, the fibres terminating in this situation. The cells of Deiter's nucleus resemble in size and shape the large cells found in the lower part of Clarke's column, and measure 60μ—90μ in their long axis. Schwalbe states that they are multipolar, and gives the measurement in their long axis as 60μ—100μ. When these cells first begin to appear in the ascending root of the auditory nerve they are mingled with smaller bi-polar cells belonging to the cuneate nucleus. The cells of Deiter's nucleus do not appear to me to be more multipolar in their character than many of the cells in the lower end of Clarke's column (*vide* micro-photo. 2).

If these cells, which closely resemble the large cells of Clarke's column, are connected with the deep fibres of the audi-

[1] *Loc. cit.*

[2] Meeting of the Physiological Society at Edinburgh, July, 1890.

tory nerve coming from the semi-circular canal, and serve as a station for the impulses transmitted from the semi-circular canals to the cerebellum, we may consider that impulses connected with equilibration are brought from the skin, joints, and tendons by fibres of the postero-external column to the cells of Clarke's column from the lower extremities, and conveyed thence to the cerebellum by the direct cerebellar tract. Impulses also may be conceived to come from similar situations in the upper portions of the body and to pass to the "cuneate nucleus," of which the cells serve as a station for impulses through the restiform body.

Organic sensations from the viscera may also be transmitted, as suggested by Dr. Ferrier, through the cells of Clarke's column to the cerebellum. All the evidence certainly goes to prove that these cells serve as a station for afferent impulses between the peripheral nervous system and the cerebellum. Only one histological fact of a doubtful nature shows that the cells of this column may be connected with the cells of the anterior horn.

There are two other groups of cells found in the spinal cord, which in shape and form differ essentially from the anterior cornual cells. These have been designated by Schwalbe *the intermedio-lateral* and *solitary*. The former are found throughout the dorsal region. The cells are bi-polar, and in vertical sections they are often seen to exist as little groups or nests of vesicular cells, from eight to twelve in number. These cells measure on an average 30μ, have their long axis placed vertically, obliquely or horizontally, and closely resemble the smaller cells of Clarke's column. In transverse sections, as also in vertical sections, may be recognised not merely the cells previously described, but also two other sets of cells of a broad spindle shape, placed usually with their long axis horizontal to the transverse section. The small ones, average size 20μ, closely resemble in form and size, and are probably identical with, the so-called solitary cells. This appears to be also the opinion of Sherrington. The larger measure 25μ-30μ, and are probably identical with the cells placed with their axis oblique or vertical. The small spindle-shaped cells attain their full size, or nearly so at birth.

As in Clarke's column, so in this tract we find many cells lying out in the lateral column. The cause of this, together with the mingling of cells of different forms to such an extent, is probably due to the growth inwards of the fibres of the posterior roots, splitting up the cells of the grey matter, which lie posteriorly and laterally to the central canal of the spinal cord during its development n embryo, and also in the early years of post embryonic life, when vertical commissural connections of the cells at different levels have to be established, together with the growth downwards of the pyramidal tracts.

These fibres, both before and after birth, cut off small portions of grey matter, and so the cells become isolated and outlying. I have never seen them lying in the still undeveloped pyramidal tract of the fœtus. I do not make any positive assertion concerning these outlying cells, as I am at present engaged in investigating the matter.

Examination of a series of sections of the spinal cord of a two-years-old child for the determination of the position of the small ovate and spindle-shaped cells situated in the lateral horns:—

1st dorsal.—A few cells.
2nd ,, Very numerous ovate and spindle-shaped cells; many placed long axis vertically, 20-30 each side.
3rd ,, Ditto, 11-15 each side.
4th ,, ,, ,, ,,
5th ,, Lateral horn well developed; cells, large and small, 14-16 each side.
6th ,, Well marked, 15-20 each side.
7th ,, ,, ,, 12-15 ,,
8th ,, ,, ,, 15-20 ,,
9th ,, Numerous cells ,, ,,
10th ,, ,, ,, 10-15 ,,
11th ,, Not so numerous.
12th ,, ,, ,,
1st and 2nd lumbar.—Some cells.

At the second sacral, as shown in fig. 4, some small ovate bi-polar cells were discovered in the sections. Too much reliance, however, cannot be placed upon this fact, because a large number of sections were examined in order to see if cells existed in this region which could, from their size and simple form, be associated with the small fibres which Gaskell has shown to emerge from the spinal cord in this region. The medium-sized bi-polar cells found

in the intermedio-lateral tract correspond in form and size with the cells of the vagus nucleus, and it may be considered that the smaller cells give origin to the fine fibres which pass out of the anterior roots throughout the dorsal region. This certainly seems much more probable than that they should come from the cells of Clarke's column. Throughout the dorsal region at the base of the anterior horn are found numerous small angular and spindle cells. These are found also in the upper cervical region, and probably are connected with fine efferent fibres.

Fine root fibres enter the posterior cornu on the outside, although they cannot be traced directly into the small bi-polar cells found at the base of the posterior cornu, and in the intermedio-lateral tract it is quite possible and probable that these small fibres find their station in these small cells, which are placed, with their long axis, horizontally, so that we might consider one pole of the cell to be connected (though not directly) with an organic sensory fibre, the other pole being continued forwards and outwards as a fine efferent fibre. Fine fibres certainly can be seen in vertical sections passing out through the lateral column, as represented in the diagram. Owing, however, to the cells being usually cut across it is difficult to trace the fibres any distance, because they tend to run forwards and outwards (*vide* fig. 3). In vertical sections of the lower dorsal regions large coarse fibres can be seen running across the lateral column. These are probably going to the direct cerebellar tract.

With regard to the larger cells resembling those of Clarke's column found in the intermedio lateral tract, with their long axis placed vertically, it is possible that these may be connected with fibres of the ascending antero-lateral. I think that the inter-medio lateral probably contains cells belonging to different systems, those placed vertically being connected with an ascending set of fibres, antero-lateral, those placed horizontally with efferent root fibres.[1]

Moreover the development of the spinal cord supports the view that the intermedio-lateral tract gives origin to the splanchnic efferent fibres of the anterior roots. He has shown that there are two kinds of efferent fibres, somatic or ventral, and lateral or splanchnic, and these take origin from entirely different parts of the basal lamina of the neural tube. The former originating from groups of neuroblasts, near the middle line, the latter near the junction of the basal with the

[1] Bechterew, '*Uber den Nerven Wurzeln*,' *Archiv. für Anatomie*, 1887.

alar lamina. In connection with the origin of the cranial efferent nerves, Schäfer states that :—

"They are disposed in two longitudinal series, one of them comprising the nuclei of origin of the somatic efferent nerves of Gaskell, which correspond with the largest fibres of a typical anterior spinal root, and the series of nuclei is a continuation of the cell column of the anterior horn. The nuclei of this series are the 12th, the 6th, 4th, and 3rd. The other series comprises the nuclei of origin of the splanchnics, efferent nerves of Gaskell, which correspond with the medium sized and smaller fibres of a typical anterior spinal root, *and the series is a continuation mainly of the cells of the lateral cornu of the intermedio lateral tract*, and partly perhaps of the cells of the base of the posterior horn. The nuclei of this series are those of the spinal accessory, those of the efferent fibres of the vagus, glossopharyngeal, facial and motor nucleus of the 5th."—QUAIN, vol. i, part 1.

Lying in the grey matter of the base of the posterior cornua, especially on the outer side, are numbers of bi-polar cells of a spindle shape placed with long axis horizontal, and with processes directed forwards and backwards—the so-called *solitary cells*.

In conclusion, although many writers have stated that Dr. Gaskell has *proved* that small efferent fibres of the anterior roots of the dorsal region are connected with the cells of Clarke's column, it is as well to state that this eminent authority did not exclude the possibility that they originated from cells of the inter-medio lateral or the solitary, which I have endeavoured to show is much more probable. Dr. Gaskell was under the impression that the cells of Clarke's column are small—hence the inference that small fibres are probably connected with small cells; and inasmuch as the distribution of Clarke's column corresponds to the outflow of fine fibres, it was natural that he should have leant to the view that Clarke's column was their source rather than the other groups mentioned by him. Gaskell[1] has recently referred to the cells of Clarke's column as divisible into two groups of different sized cells. Sherrington, in a footnote concerning this point, makes the following statement :—"I must confess, however, I have not been able to convince myself of the division of the cells

[1] 'Journal of Physiology,' vol. x., p. 157, 1889.

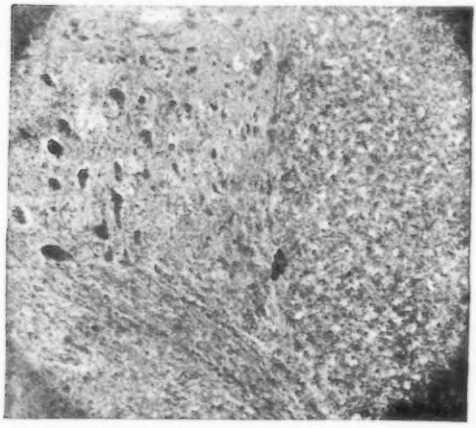
into two groups, nor do I interpret the observations of Mott[1] to be in support of the division of cells into two groups." I think I can explain this discrepancy.

Schwalbe[1] has pointed out in connection with the anterior cornual cells, that where a column is well developed the cells are larger—therefore the cells of the lower end of Clarke's column are larger. Moreover, owing to their connections with the fibres of the postero-external column on the one hand, as shown in fig. 2, and on the other hand with the fibres of the direct cerebellar tract, the cells are placed not with their long axis vertical, but either obliquely or horizontally, so that they appear in transverse sections much larger here than higher up. Consequently in transverse sections of the lower end of the column one sees the long axis of the cell, whereas higher up one sees the vertically-placed bi-polar cell cut transversely, and, therefore, only the narrower transverse axis is measured. I also stated that in the postero-external column were two sets of fibres, large and comparatively small, as shown in micro-photo. 2. The former I believed to be connected with the larger cells of Clarke's column, the latter with the smaller. This may be so, but still the impulses passing through the cells would be afferent. From experimental observations I am more inclined to believe that these smaller fibres belong to Goll's column, being on their way there through the postero-external column. This much may certainly be said, that the smallest cells I have seen in Clarke's column, under any circumstances, have been larger than the largest cells found in the other two groups. This fact, when coupled with the fact that at birth the cells of Clarke's column are only one-third of the size of the cells at two years, strongly suggests that they are not connected with fine efferent fibres having an organic function such as the innervation of the visceral-vascular system.

Moreover, the whole of the cells of the spinal cord would be absorbed for efferent fibres, according to Dr. Gaskell's theory, leaving no cells for two distinct afferent tracts to the brain, viz.,the direct cerebellar and antero-lateral, which have been shown not to have their origin in the ganglion cells of the posterior root.

[1] *Loc. cit.*

TO ILLUSTRATE DR. MOTT'S PAPER ON THE BI-POLAR CELLS OF THE SPINAL CORD AND THEIR CONNECTIONS.

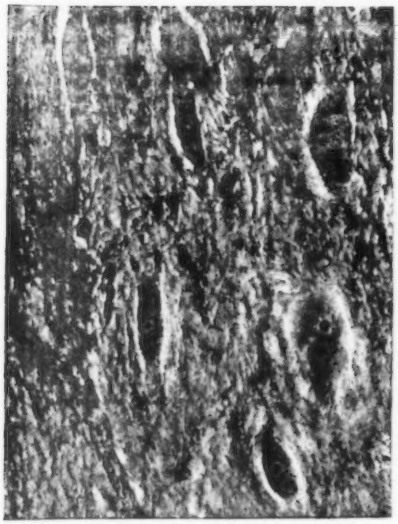


MICRO-PHOTO. I.

Transverse section of fœtal spinal cord, lower dorsal region, shewing cells of Clarke's column: magnified 150 diameters.

MICRO-PHOTO. II.

Transverse section of Clarke's column in the lower dorsal region (11th-12th segment), showing many large cells: and, in the fibrillary net work, large fibres are seen cut transversely. Magnified 250 diameters.



MICRO-PHOTO. III.

Vertical section of Clarke's column shewing the large bi-polar cells lying in a spongy network of nerve fibrillæ. In the original photograph the fine fibrils can be seen passing across the lymph space to join the cell. Magnified 250 diameters.

MICRO-PHOTO. IV.

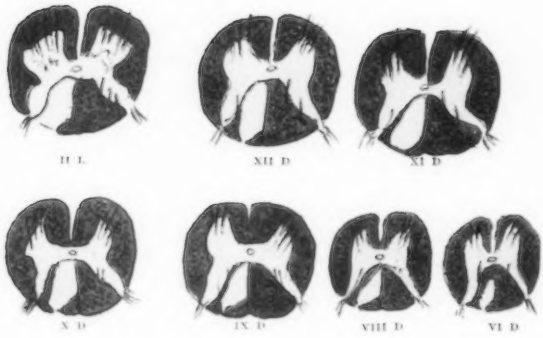
Spinal cord of monkey at 12th dorsal. The seat of the lesion is seen on the left side. Destruction of nearly the whole of the grey matter on that side, and shrinking of left half of the spinal cord.

MICRO-PHOTO. V.

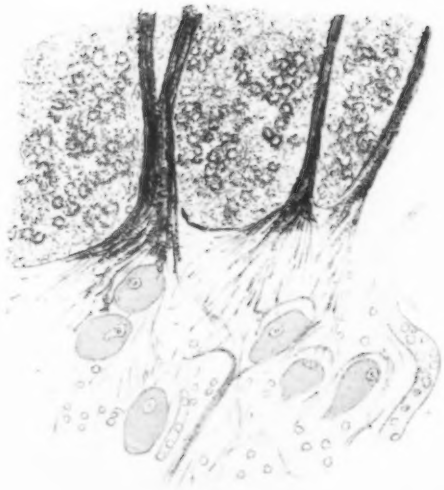
Clarke's column from the previous section, as seen under a magnification of 150 diameters. On the right hand side of the central canal are seen the cells lying in a fibrillary network; on the left, disappearance of the cells and atrophy of the network.

MICRO-PHOTO. VI.

Portion of the lateral column in the mid-dorsal region on the side of the injury, showing the degenerated fibres, stained by Weigert's method, after

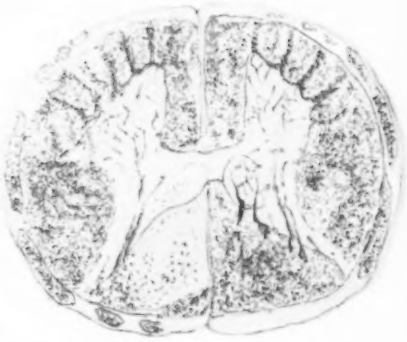


I.—Unilateral section of the roots of the Cauda Equina in the Monkey.
Mag. 4 diameters.



II.—Clarke's Column human spinal cord, 12th dorsal. Fibres from postero-external column entering and terminating in a brush of fine varicose fibrils around the cells which are for the most part cut across obliquely.—Mag. 250.

III.—Vertical section of lateral column and intermedio-lateral tract mid dorsal region, 2 yr. human spinal cord.
Mag. 250 diameters.



V.—Spinal cord of Monkey at level of 12th dorsal. Degeneration of postero-external column on left side as a result of section of 3d, 4th, and 5th lumbar and 1st and 2d sacral. Atrophy of nerve plexus around cells of Clarke's column on the same side.
Mag. 10 diameters.

IV.—Cells of intermedio-lateral seen in transverse section of 2d sacral.
Mag. 250 diameters.

THE EFFECT OF MOVEMENTS OF THE HUMAN BODY ON THE SIZE OF THE SPINAL CANAL.

BY R. W. REID, M.D., F.R.C.S.
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(*From the Physiological Laboratory, St. Thomas's Hospital, London.*)

Our object in making the following short series of experiments has been to test the possibility of an alteration taking place in the capacity of the cerebro-spinal canal during the performance of such movements as can, under ordinary or extraordinary circumstances, occur in the jointed walls of that canal between the bones composing the cranio-vertebra axis. A few preliminary observations fully sufficed to show us that a change of capacity of the canal does actually take place, although only to a slight extent. These detectable but small alterations it seemed highly desirable to measure, and obtain permanent records of. The following plan of experiment was therefore devised and in its ultimate form found successful.

The cadaver, the subject of observation, was suspended from the horizontal arm of a strong vertical stand by means of an iron coronet which encircled the skull and held it by four sharp-pointed thumbscrews, screwed into, but not perforating, the calvaria. An additional precaution against the slipping downward of the cadaver was obtained by inserting into the external auditory meatus the ends of a scissors-shaped clip (B). This clip was attached to a rope running over two pulleys at the top of the vertical support. A horizontal iron bar (C) which could be adjusted at any desired height on the vertical stand was employed to act as a fulcrum against which the body could be bent.

The procedure of experiment was as follows. The scalp over the vertex having been removed, a trepan hole measuring 35 mm. across was made in the highest part of the skull, close to one side of the superior longitudinal sinus. The dura mater was thoroughly removed to an extent corresponding with the trepan hole. We then generally found that the brain was separated from the parietes of the cavity sufficiently to admit the handle of a scalpel between it and the overlying dura mater. Water was then poured into the subdural space sufficient to fill up the trepan hole. The water introduced tended to disappear, often rapidly, sinking in level into the subdural space. Especially was this so with the first few ounces poured in; after a certain amount had been introduced the sinking became less rapid. When the trepan hole was quite filled with water a small glass tube (D) surrounded by an india-rubber ring was inserted with as little delay as possible into the hole, and was arranged to fit into it in a perfectly water-tight fashion. Over the end of the glass tube had been previously drawn a thin membrane which floated on the water filling the subdural space; this membrane was made sufficiently redundant to allow it a considerable range of movement on the water. The glass capsule itself was filled with water and put into communication by flexible water-tight joints, with a length of glass tubing almost horizontally placed (E). The farther end of this tubing was the lower, and was placed on the same level as the trepan hole in the skull. In the figure the inclination of this tube is represented as greater than it really was. Finally, this almost horizontal tube and the connections intervening between it and the vertical glass capsule were all completely filled with water.

Beneath the free end of the tube the scale-pan of a sensitive balance was placed in such a way that it received any drops of fluid ejected from the tube. To the other end of the beam of the balance was attached a thread, which passed vertically downwards, and after running round a light pulley hung, carrying a little weight and armed with a small pen arranged for writing upon the blackened surface of a recording drum. The amount of water received by the scale-pan

was represented by the height of the line marked by the recording pen. The scale-pan naturally underwent movement along the arc of a circle; the intervening pulley round which the thread passed converted the movement of the thread into a rectilinear one; in order, therefore, to estimate the amount of water in the pan the drum had before each set of observations to be experimentally graduated. In each observation the measurement recorded is that of the *maximum* displacement obtained in a series of repetitions of the particular movement.

I. *May* 8, 1889.—Cadaver of male child, æt. four years, weight 24 lbs. 10 oz. Cause of death, diphtheria. Trephined at vertex, to left of median line. Head fixed in the coronet in such a way that no part is higher than the median edge of the trepan-hole.

Body hanging freely from the head:—On raising the trunk by the pelvis, thus taking off the extension of the spinal column due to the weight of the trunk and limbs, 263 cubic millimetres of fluid were ejected into the scale-pan. On raising the trunk by the legs 255 cubic millimetres were ejected. When the body was bent far backwards 1,500 cubic millimetres ejected. When the body was bent far forwards 258 cubic millimetres ejected. When the body was rotated by the shoulders either to left or right 78 cubic millimetres ejected.

II. *October* 30, 1889.—Cadaver of male child, æt. seven years; cause of death diphtheria. Trephined at vertex to right of median line.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 450 cubic millimetres; bending the body backwards caused ejection of 530 cubic millimetres; bending the body forwards caused ejection of 215 cubic millimetres: rotation by the shoulders to right or left caused ejection of 180 cubic millimetres.

Body hanging as before but with the cervical spine fixed against bar:—Bending the body backwards caused ejection of 215 cubic millimetres; bending the body forwards caused ejection of 180 cubic millimetres.

III. *February* 14, 1890.—Cadaver of woman, æt. twenty-

eight years; cause of death, pericarditis. Trephined at vertex to right of median line. Weight of cadaver, 7 st. 4 lbs.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 98 cubic millimetres; bending backwards the body caused the ejection of 480 cubic millimetres; bending the body forwards caused ejection of 220 cubic millimetres; rotation by the shoulders right or left caused ejection of 110 cubic millimetres.

Body hanging as before but with cervical spine fixed against the bar:—Bending the body backwards caused ejection of 230 cubic millimetres; bending the body forward caused ejection of 200 cubic millimetres; bending the trunk backwards through an angle of 40° against a fulcrum two inches below the inferior angle of the scapula caused an ejection of 2,050 cubic millimetres; bending the trunk forwards through an angle of 45° over the fulcrum at same level as last gave a displacement of 20 cubic millimetres.

IV. *November* 20, 1889.—Cadaver, male, weighing 11st. 4lbs., æt. thirty years; cause of death, typhoid.

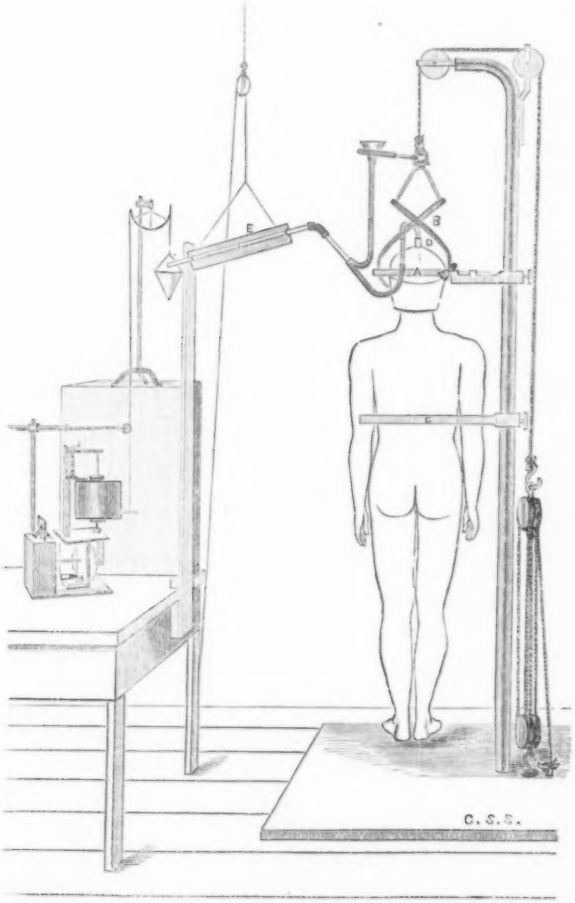
Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 190 cubic millimetres; bending the body backwards caused the ejection of 1,025 cubic millimetres; bending the body forwards caused the ejection of 600 cubic millimetres; rotation by the shoulders to right or left caused ejection of 100 cubic millimetres.

Body hanging as before, but with cervical region of spine fixed against the bar:—Bending the body backwards gave ejection of 450 cubic millimetres; bending the body forwards gave ejection of 150 cubic millimetres; bending trunk backward through 40° over a fulcrum two inches below inferior angle of scapula gave ejection of 2,400 cubic millimetres; bending trunk forward through 45° over fulcrum at similar level gave ejection of 28 cubic millimetres.

V. *November* 16, 1889.—Cadaver, male, powerful frame, æt. thirty-six years, weight 11st. 2lbs.; cause of death, pneumonia.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 70 cubic millimetres;

TO ILLUSTRATE PAPER ON THE EFFECT OF MOVEMENTS OF THE HUMAN BODY ON THE SIZE OF THE SPINAL CANAL.



bending the body backwards caused the ejection of 1,800 cubic millimetres; bending the body forwards caused the ejection of 920 cubic millimetres; rotating the shoulders to right or left caused ejection of 100 cubic millimetres.

Body hanging as before, but with the cervical spine fixed against the bar:—Bending the body backwards caused ejection of 800 cubic millimetres; bending the body forwards caused ejection of 260 cubic millimetres; bending trunk backwards through 40° over fulcrum two inches below inferior angles of scapula gave ejection of 1,250 cubic millimetres; bending forward through 45° over fulcrum at same height gave ejection of 30 cubic millimetres.

VI. *November* 15, 1889.—Cadaver, male, weighing 12st. 4 lbs. Cause of death, cardiac disease.

Body hanging freely from the head:—Raising the trunk by the pelvis caused ejection of 48 cubic millimetres; bending the body backwards caused ejection of 1,200 cubic millimetres; bending the body forwards caused ejection of 250 cubic millimetres; rotation by shoulders to right or left caused ejection of 140 cubic millimetres.

Body hanging as before, but with cervical spine fixed against the bar:—Bending the body backwards gave ejection of 900 cubic millimetres; bending the body forwards gave ejection of 200 cubic millimetres; bending body backwards through 40° over fulcrum two inches below inferior angle of scapula, gave ejection of 2,300 cubic millimetres; bending body forward through 45° over fulcrum at similar level gave ejection of 36 cubic millimetres.

VII. *October* 22, 1889.—Cadaver, female; weight, 7 stone 10 lbs.; æt. fifty-four years. Trauma.

Body hanging freely from the head:—Raising the trunk gave ejection of 90 cubic millimetres bending the body backwards caused ejection of 280 cubic millimetres; bending the body forwards caused ejection of 145 cubic millimetres; rotating the shoulders to right or left gave ejection of 95 cubic millimetres.

Body hanging as before but with cervical region fixed against bar:—Bending body backward gave ejection of 170 cubic millimetres; bending body forward gave ejection of

40 cubic millimetres; bending trunk backward through 40° over fulcrum at inferior angle of scapula gave ejection of 1,100 cubic millimetres; bending forwards through 45° over similar fulcrum gave ejection of 32 cubic millimetres.

QUANTITY OF FLUID DISPLACED FROM CRANIO-VERTEBRAL CANAL DURING MOVEMENTS OF THE BODY.

I.—Cadaver Hanging Freely from the Head.

Age in Years.	Sex.	Relieving the spine from the weight of the body by lifting.	Bending Body.		Rotation by the shoulders.
			Backward.	Forward.	
		Cub. Mil.	Cub. Mil.	Cub. Mil.	Cub. Mil.
4	♂	263	1500	258	78
7	♂	450	530	215	180
28	♀	98	480	220	110
30	♂	190	1,025	600	100
36	♂	70	1,800	920	100
45	♂	48	1,200	250	140
54	♀	90	280	145	95

II.—Cadaver Suspended from Head, but with Cervical Region of the Spine Prevented from Bending by a Bar.

Age in Years.	Sex.	Bending Backward.	Bending Forward.	Bending of spinal column over a bar at level close below the inferior angle of the scapula.	
				Backward.	Forward.
		Cub. Mil.	Cub. Mil.	Cub. Mil.	Cub. Mil.
7	♂	215	180	—	—
28	♀	230	200	2,050	20
30	♂	450	150	2,400	28
36	♂	800	260	1,250	30
45	♂	900	200	2,300	36
54	♀	170	40	1,100	32

Conclusions that we arrive at from our observations may be shortly set forth as follows :—

1. That when the body hangs freely and vertically from the skull the capacity of the cranio-vertebral canal is at a maximum.

2. That with the body in the above position, when the weight of the trunk and limbs is taken off by, for instance, lifting and supporting the body vertically, there is a diminution in the capacity of the cranio-vertebral canal, but the diminution is a very slight one.

3. That when the vertebral column is bent backward or forward, especially in the former direction, there is a not inconsiderable diminution in the capacity of the cranio-vertebral canal as compared with its capacity when the body is hanging freely and vertically.

4. That the alterations in the curvatures of the spinal canal by various movements of the body do influence the capacity of that canal, but not to any great extent; much more however in the child than in the adult.

A point of practical interest may be adverted to. From the above measurements it would appear that by suspension the size of the spinal canal is increased in the adult of middle age to the extent of some 100 cubic millimetres. Estimating the total capacity of the canal at 102 ccm., this measurement means an increase in the size of the canal to the extent of 1-1020·0th of the whole canal. This increase is so small that it becomes difficult to conceive how, in the "suspension" treatment lately introduced for cases of tabes dorsalis, &c., there can, as has been sometimes claimed, be any actual stretching of the spinal cord.

Clinical Cases.

NOTES ON THREE CASES OF AN HEREDITARY FORM OF PROGRESSIVE AMYOTROPHY.

BY H. B DONKIN, M.B.OXON, F.R.C.P.

Physician to Westminster Hospital, and to East London Hospital for Children.

THE cases described below were shown by me at the Neurological Society, in June, 1890, and were considered by most observers to be fitly classed with those described by Leyden in 1875, in which the symptoms are noted as beginning in the lower extremities. In 1884 Dr. Ormerod[1] published three very similar cases in one family under the heading of "cases of muscular atrophy after measles." In 1886 MM. Charcot and Marie[2] described an affection of this kind; in the same year Dr. Tooth[3] dwelt on the subject at length, with cases, under the title of the "Peroneal Type of Muscular Atrophy." His papers are the English *locus classicus* of this matter up to the present time. Dr. Herringham[4] gave an important and detailed account of one case in 1888.

CASE I.—Walter Rivitt, aged nine years, admitted into the East London Hospital for Children in June, 1890, for difficulty in walking. He is a healthy-looking boy, and there is no evidence of any disease other than is mentioned below. (The fundi oculorum are normal.) His gait is high-stepping, not spastic; the cause being foot-drop owing to weakness of muscles of dorsal flexion, so that the leg has to be raised high for the toes to clear the ground. The feet are in the position of partial varus, both at rest and in walking. This is most marked on the left side. The absence of any contraction shows that it is due to peroneal weakness. In walking the ball of the foot is brought down suddenly on the ground before the heels. All the muscles of the legs below the knees are evidently wasted, while the thigh muscles are appa-

[1] BRAIN, Oct., 1884. [2] *Revue de Médecine*, Feb., 1886.
[3] BRAIN, vol. x., p. 243, and *St. Bartholomew's Hospital Reports*, vol. xxv.
[4] BRAIN, July, 1888.

rently normal. Sensibility to pain, as tested by pricking, seems to be decidedly diminished over the leg-surface, and a faradic current causing great pain elsewhere is here said to be unfelt. Similar insensibility is apparent even after a prolonged application of the negative electrode of a galvanic battery. There is no reaction to faradism or galvanism in any of the muscles below the knees, while the thigh-muscles react quite normally. The knee-jerks are present, and there is no ankle-clonus.

The upper extremities show the ordinary marks of progressive muscular atrophy affecting the hands; there is a moderate degree of the *main-en-griffe*, and considerable wasting of the thenar and hypo-thenar muscles. There is the same apparent loss of sensibility over the skin of the hands, and there is no reaction to faradism or galvanism in any of the short muscles of the hands, except perhaps very slightly in the first and fourth dorsal interossei. The forearm muscles respond less readily to faradism on the extensor than on the flexor aspect. There is no reaction of degeneration anywhere, and the muscles of the rest of the body react normally in every respect.

This boy's *personal history* was that the weakness in the ankles began two years before admission, very soon after an attack of measles, and has lately become rapidly worse. The hands were first noticed to be weak about six months ago. He had measles twice, the weakness following on the second attack; chicken pox and inflammation of the lungs when very young; no fits or other illness. The mother states that he has always been intelligent and very healthy. He seems perfectly intelligent now.

The *family history* is that the father, a carman, is healthy, but walks like the patient, and has "contracted hands." The mother is healthy. She had eight children; two were still-born, two died of "fits" in infancy, and of the four living a sister, aged seventeen, suffers like her brother and father. The mother had two miscarriages after the patient's birth. There is no history or evidence of syphilis in any of the family. The patient's paternal great-grandfather "suffered in his hands" in the same way. No further history of nervous or other disease.

Case II.—Alice Rivitt, the above-mentioned sister, aged seventeen years, came to the hospital at my request, and was examined by Dr. Hastings, the resident medical officer. The gait and wasting are almost exactly the same as in her brother's case, but the wasting is more marked in the upper extremities. The wasting is said to have begun in the legs at the age of seven (as in the brother's case) and also just after a second

attack of measles. The wasting of the hands was observed two years later. For several years now she has not been getting worse in any respect.

The knee-jerks are present, and there is no ankle-clonus; there is loss of sensibility of the same distribution as in the brother's case. The muscles below the knees fail to react; the thigh muscles react, but less readily than the brother's. In the right forearm the long extensor of thumb and both carpal extensors react, but no short muscles. In the left forearm all extensors of thumb and wrist react, but imperfectly; the extensors of fingers do not react. All flexors of forearm except those of thumb react, but none of the short muscles. The muscles of both upper arms react somewhat imperfectly, but all others are normal. No fibrillar or other tremors have been observed in either of the patients.

Case III.—The father of the above two patients, æt. forty-two, was also examined electrically by Dr. Hastings. He stated that he was about seventeen when the weakness appeared in his legs, and that the hands began to suffer in the same manner as those of his children about two years afterwards. He does not remember any previous illness. For many years he has been as bad as he is now, with marked deformity of feet and hands. On the left side the leg below the knee is very much wasted, all the muscles being affected, but especially the anterior tibial and the peronei. The foot is in the equinus or slightly equino-varus position, and is much hollowed out on the inner side. There is no power of flexing the ankle, but he can extend it after passive flexion. There is no movement of the toes. The right leg is similar, but the wasting is everywhere less, and the muscles have more tone; the movements, however, are but very slightly better. Both upper arms are well nourished, and their movement normal.

To faradism the extensors of right forearm react, but the E. carpi ulnaris less so than the rest; both flexors of the wrist act, but none of the short muscles of hand to either current. On the left side all the forearm muscles act normally, but none of the hand muscles. Below the knees there is no reaction to either current, and the patient does not feel any pain on the application of the electrodes. The left trapezius muscle shows a slight degree of R.D. The other muscles are normal. The knee-jerks on both sides are very brisk, and there is nc ankle clonus. No tremors anywhere, and no marked loss of sensibility to pricking. The man underwent tenotomy for his feet at the age of twenty as a surgical patient at one of the London hospitals.

Remarks.—These cases show the closest resemblance to Dr. Ormerod's, which were also of a father and two children. All six began in early life, the electrical reactions were practically the same in all, and there is a history of measles immediately preceding the affection in five. The father in Dr. Ormerod's cases was, however, affected in one leg only, and from this fact and the want of opportunity of complete examination, Dr. Ormerod hesitated to pronounce a definite diagnosis in this instance.

In Dr. Herringham's case there were fibrillar tremors, no apparent alteration of sensibility, and only diminished reaction to faradism, with no R.D.; but many features of the case seem to be the same. Dr. Herringham traced the disease through five generations, the transmission appearing to take place through females to males alone.

Dr. Tooth's collection of cases shows that heredity, though frequent, is not always present. He also mentions marked pains in some instances, and tremors, with faradic contractility and unaltered galvanic reactions in the early stage.

It must be remembered that Dr. Ormerod's and my own cases, where there was no reaction to either current in the affected muscles, were mostly of many years' standing; yet two of them, boys of nine years old, showed this characteristic after th. disease had lasted two and four years respectively.

It seems probable that the occurrence of measles played the part of an immediately exciting cause, or occasion, of the neuro-muscular breakdown which marks these cases. There appears to be no definite characteristic by which either a myelopathy or a peripheral neuropathy or a myopathy can be established or excluded as the original morbid process. The absence of fibrillar tremors probably points *per se* to a myopathy, but there is a want of observation on this point in the early period of the disease. The marked absence of reaction to both currents, which is only known with certainty as obtaining in the advanced stages of the disease, does not give much aid in localizing the original lesion. Whether all the cases I have referred to are to be regarded as coming under one general category can only be settled after more knowledge has been gained from clinical observation of the affections at their outset. Further research on this point, and perhaps fresh facts supplied by morbid anatomy, may possibly in time throw some light on the vexed question of the neuropathic or myopathic origin of any, or all, of such cases—if, indeed, that question be a soluble or scientific one at all. In ultimate ætiological analysis it is

probably a vain and merely scholastic speculation to inquire as to the exact point where neuropathy and myopathy meet or depart, clear though the difference may be between centrally and peripherally-caused neuro-muscular affections as a whole.

The above cases bear some general clinical likeness to pseudo-hypertrophic paralysis, and their exact pathology is about equally obscure.

A CASE OF RECOVERY FROM CEREBRO-SPINAL MENINGITIS.

BY W. HALE WHITE, M.D.
Physician to Guy's Hospital.

JAMES CLEMENTS, æt. 19, living at 15, Earls Road, Old Kent Road, a railway servant, was admitted into John Ward, Guy's Hospital, 29th June, 1889, complaining of severe pain in the back of the head. His mother has paralysis agitans, and his sister is said to have fits.

He first felt ill 27th June, 1889, when he became giddy. He has vomited several times since, and has complained of headache. There is no history of any disease of the ear or nose, nor of any blow.

On Admission.—The head is kept rigidly fixed in the middle line, and is slightly retracted. There is much headache and considerable pain and tenderness over the upper cervical spines. He does not not move his trapezius when he attempts to shrug his shoulders. Pulse varying in rapidity, rarely more than fifty-eight, often a little less, irregular, occasionally it intermits. The pulse and temperature have lost their mutual relation, thus with the pulse at fifty-eight the temperature was 102° F. Respiration irregular, twenty-two to the minute. Sometimes the diaphragm alone is in action, at other times only the costal muscles, the two rarely acting together. Breath sounds normal.

The tongue is coated with a slight fur. There is little appetite, the bowels have not been open since June 26th. The urine is healthy.

Nervous System.—The patient is delirious at times; he complains of great headache. He says the limbs are weak, but the grasp is fairly good. There is no obvious paralysis in any part of his body, but certain movements of the head cause pain. There is no anæsthesia nor hyperæsthesia. The lower edge of the right optic disc is slightly blurred, and the whole surface is redder than normal. The left optic disc is much blurred and red. There is

photophobia. The pupils are equal and react normally. The superficial and deep reflexes are all normal.

Ice bags were applied to the head and neck. A single dose of a minim of croton oil was given. The patient was put upon moderate doses of Hydrargyrum cum Creta, which was continued during the whole of his stay in the hospital. Milk diet was ordered.

June 30th.—Patches of herpes have appeared round the mouth on the left side only; there are no signs in the lungs to account for this. All the symptoms remain the same, except that the fixation of the neck and the pain are not quite so severe.

July 3rd.—The symptoms are the same, except that in every respect the patient seems much better.

July 4th.—The patient is worse again. He is delirious, he mutters continually, and he allows flies to collect on his face. The breathing is very irregular. He passes his urine and fæces under him. The optic discs remain the same as on admission. It is difficult to tell whether he has much pain. The pulse is slow and irregular. He vomits a good deal, and the vomiting is characteristically cerebral. Although the patient is so much worse, the temperature is only 98·8 F.

July 5th.—Is in much the same condition as yesterday.

July 6th.—Is worse in every respect, save that the temperature remains normal. The symptoms are unchanged, but there is in addition decided external strabismus in the left eye.

July 10th.—Since the last note the patient has been improving in every respect. He is now rarely delirious; the headache is better. He is very weak, but takes milk well. The temperature is about normal.

July 12th.—This morning there is a change for the worse. The patient is unconscious, he has muttering delirium, and passes everything under him. He takes no notice of anybody nor anything. He still has strabismus. His pulse is irregular and slow.

July 17th.—During the last five days the patient has been so ill that it has always seemed almost impossible that he can survive more than a few hours. The symptoms have remained the same as since the last note, except that they are much worse, and he has been much troubled with hiccough.

July 19th.—For the last two days the patient has been much better. He has become conscious, and no longer passes his urine and motions under him. He is as thin and wasted as any patient I have ever seen. From this date he gradually improved, very slowly at first but more rapidly afterwards, so that by August

20th he was gaining 2lbs. a day. All his symptoms disappeared before he left the hospital at the end of September, except the strabismus.

Nov. 21st, 1889.—The patient was seen to-day. He says he can walk any distance, and he has gone back to his work as a truck shunter on the railway. Since he left the Hospital his weight has increased from six and a half stone to ten stone. The only thing he complains of is that he occasionally gets a pain down the back, but this trouble is not severe. He has no headache. The optic discs are perhaps a trifle red, but they are hardly abnormal. The squint that he had when he left the Hospital has disappeared, but the movements of the eyes are not always perfectly free. The pulse is regular. It is interesting to note that he has a slight patch of herpes on the lip in the same place as the herpes broke out when he was in the hospital. His knee reflexes can only be obtained with difficulty. He is quite sure he never had herpes before this illness.

January 17th, 1890.—Re-admitted into Guy's Hospital under Dr. Taylor. About a month ago he began to notice that his right leg was getting weak; this has gradually become worse. His right arm has been getting weak for the last ten days. Since his discharge he has never suffered from headache or sickness, nor has he had any fits.

Condition on admission.—Heart, lungs, urine, and temperature all normal. He looks healthy. He is very emotional, weeping about the loss of power in his leg. Intelligence and memory good.

Sensation.—There seem to be slight impairment and delay of sensation all over right limbs and the right side of his body. There is no analgesia, nor paræsthesia. Hearing, sight, and optic discs normal. Muscular sense for arms the same on the two sides. Differences of 1 in 13 are rightly distinguished.

Motion.—Superficial reflexes normal, no knee jerks, no ankle clonus. On smiling, the right side of the face moves slightly less than the left, and the tongue is protruded slightly to the right. Movements of respiration normal. Loss of power in both arms, especially the right. With the dynamometer the right grasps 50, the left grasps 60. No difference in measurements, no rigidity. Loss of power in both legs, especially the right.

Right thigh, 16 inches in circumference.
Left ,, $15\frac{1}{2}$,, ,,
Right calf, $10\frac{3}{4}$,, ,,
Left ,, 12 ,, ,,
Left gluteus mamimus seems smaller than right.

The loss of power is most marked in both anterior tibial muscles, and in the right rectus. His gait is very unsteady, but it is not characteristic of any particular form of disease. The electrical reaction of all the muscles are normal, except that in the flexors of the right forearm, ACC>KCC.

He went out on February 18th, 1890, having in all respects rather deteriorated since admission.

During the summer of 1890 he was for some time in the hospital at Queen's Square.

Early in October, 1890, I saw him. He was still weak in his legs, and his gait was staggering. He was also lachrymose.

November 20th, 1890.—I saw the patient to-day, he is now much better. There is no ocular paralysis; both the legs and arms are rather weak, but he can walk a long distance, although his gait is still a little unsteady. His knee jerks are normal. He does not complain of any sensory symptoms, but he is inclined to cry. On the whole he is much better.

PARALYSIS AGITANS IN A YOUNG MAN.

BY W. B. HADDEN, M.D., F.R.C.P.

THE patient was a man, aged twenty-eight, a shoemaker by trade, who was sent to me by Mr. A. L. Perkins, of Sketty, South Wales.

There was nothing of importance to note in the family history.

When two years old he had an attack of infantile paralysis, and when fifteen years of age he had typhoid fever. When he was twenty-five he had an attack of what, from his description, would appear to have been acute rheumatism. Three months later the left hand began to tremble, and since the onset the movements have gradually become worse.

The patient was a healthy-looking man, with a fixed expression of face. There was no marked defect of articulation, but when he spoke there was little movement of the face beyond the immediate neighbourhood of the mouth. This was made especially evident by covering up the mouth and chin, and then getting him to speak. The body and head were not inclined forward, as is often the case in paralysis agitans. There were no tremors of the head or tongue, and no nystagmus. When directed to look to one or other side the movements of the head and eyeballs were synchronous. The left upper extremity was slightly flexed at the elbow, and the forearm in a position midway between pronation and supination. The whole limb was affected by rhythmical movements of abduction and adduction. The metacarpo-phalangeal and phalangeal joints were slightly flexed, and the digits were the seat of constant to-and-fro movements, which were much increased when he was excited, or when he was being watched. There was alternate flexion and extension of the wrist simultaneous with the movements of the fingers. When on the point of taking a glass of water the movements diminished for the moment, but increased again when the glass was seized, and became very marked as it was carried to the mouth. The movements ceased entirely during sleep. With the

left hand he was slow in performing voluntary movements, such as buttoning and unbuttoning, and in threading a needle (he was a shoemaker). There was no coarse muscular weakness, and no loss of sensation of the affected limb. There were no movements elsewhere in the body. The right lower limb was considerably wasted, though no special muscular groups were picked out. There was double pes valgus; the knee-jerks were feeble. When walking he waddled from side to side, and limped with the right leg, but his gait was not slow, and there was no tendency to festination.

Remarks.—I have no doubt the case was one of true paralysis agitans. I had intended showing the patient at the Neurological Society, but when he found that treatment was without effect he declined to stay longer in the hospital. My friend, Dr. Beevor, saw the man, and confirmed the view which I had taken. The onset of paralysis agitans at the age of twenty-five is very exceptional, but a few cases have been recorded even at an earlier age.

Critical Digests.

FRIEDREICH'S DISEASE.

BY DR. P. LADAME.

(Read before the Medical Society of Geneva. Translated by W. Dudley, M.B.)

DUCHENNE de Boulogne* had just published his classical study on progressive locomotor ataxy (1858 and 1859). This work aroused great interest, and immediately gave rise to numerous controversies, of which we still hear the last echoes. Duchenne has often been reproached, and with reason, with having caused an unfortunate confusion by this name, "locomotor ataxy," from his taking the symptom for the disease. Jaccoud† one of the first, in a historical critique too severe to be fair, accuses Duchenne of having fallen into an error, carefully avoided in recent times, when he named this disease from the functional trouble, from the symptom which it causes. "It is," says Jaccoud, "a nosological error of the same kind as if one called pulmonary phthisis, chronic cough; pneumonia, acute cough; or myelitis, paraplegia."

Nothing discloses this error better than the history of the disease described for the first time by Friedreich, September 18th, 1861, at the Congress of German naturalists and physicians at Spire. The eminent clinician of Heidelberg had indeed observed with the greatest care several cases of locomotor ataxy, the symptoms of which differed greatly from those which had been described two years before by Duchenne in the *Archives générales de Médecine*. Now Friedreich, who frequently had the opportunity, in

* DUCHENNE DE BOULOGNE, De l'ataxie locomotrice progressive. *Arch. gén. de méd.*, Dec. 1858, Jan., Feb., April, 1859.

† S. JACCOUD, Les Paraplégies et l'Ataxie du mouvement. Paris, 1864, p. 583

1858 and 1859, of showing his cases to several of his colleagues, among others to Eisenmann, Hasse, Kussmaul and Virchow, accepts only with reserve the clinical picture of Duchenne. He protests even against the pathological type created by the latter. "The complexus of symptoms," says he, "which constitutes progressive locomotor ataxy of Duchenne, cannot absolutely be considered as representing a distinct disease *sui generis*. It is a morbid entity, a group of symptoms which has its anatomical and physiological characteristics, but it is not a special disease, for progressive locomotor ataxy, the failure of co-ordination of voluntary movements, presents very great variations." Witness the cases observed and described by Friedreich, which offer very remarkable differences from those of Duchenne.

Friedreich, with his clinical insight, had therefore correctly guessed that his cases did not come under the description of locomotor ataxy of Duchenne. He did not think, however, that he ought to separate them entirely. He thought that the picture of Duchenne was incomplete, and as one of the fundamental differences between his cases and those of the great French pathologist was found to consist in the absence of sensory disturbances, he adopted the following hypothesis, which might, according to him, furnish a satisfactory explanation of it.

"If, therefore," says Friedreich (3), "in the different cases of locomotor ataxy which have been published, we find sometimes sensory disturbances, sometimes complete absence of them, it is because, in the first case, degeneration of the medullary fibres is not limited to the posterior columns, but has invaded other regions of the spinal cord."*

When he put forward this hypothesis, ingenious rather than well founded, Friedreich had already made three autopsies, the results of which would have permitted him to conclude that there might be combined sclerosis of the posterior and lateral columns, since this lesion manifestly existed in two of his cases.

Nevertheless, many years will pass before the ataxy of

* JACCOUD, *loc. cit.* p. 677, adopts this erroneous hypothesis.

Friedreich takes rank in nosography. For a long time, quite recently even, we have seen it, this morbid form will be considered by the authors, Topinard, Erb, Eulenburg, Jaccoud, Möbius, Grasset, Strümpell, &c., as a variety of tabes dorsalis (locomotor ataxy of Duchenne). To-day even, it is in the chapter on this affection that we must look for the description of *hereditary ataxy* in pathological works.

? Hereditary ataxy! That is, indeed the name chosen by Friedreich as the title of his second memoir. The disease is not observed, he thought, in isolated individuals. Each time that he had seen a case of it he met with several others in the same family amongst the brothers and sisters of the patient. Eisenmann,† who speaks nowhere of Friedreich's cases, was the first to employ the words hereditary ataxy (*erbliche ataxie*), quoting as an example the case observed by Carre, and the opinion of Trousseau. This name of hereditary ataxy has been justly criticised. M. Féré ([33]), following Dr. Gowers ([25]), of London, and Brousse ([34]) of Montpellier, has shown that it was "manifestly wrong," since, on the one hand, classical locomotor ataxy recognised also very frequently for primary cause, either direct heredity, or more often neuropathic heredity, and, on the other hand, Friedreich's disease is absolutely distinct from locomotor ataxy, and consequently should not be confounded with it under a common name. Moreover, it is not heredity which is the essential and striking feature in this affection; it is rather its "familial" character, that is to say, the frequency of the development of the disease in several children of the same family. Dr. Everett Smith ([60]), of Boston, proposed for this reason to call it "postero-lateral spinal sclerosis of generic origin," or more simply "generic ataxy." This name, however, does not suit a disease which has nothing in common with progressive locomotor ataxy, as Pitt ([74]) says, except the abolition of the knee jerks.

After having been regarded as a particular form of locomotor ataxy of Duchenne, Friedreich's disease was also considered by some authors as a simple variety of dissemin-

† EISENMANN in Würzburg, Die Bewegungs-Ataxie. Wien, 1863, p. 205.

ated sclerosis, or at least as a combination of locomotor ataxy and multiple sclerosis. Charcot* and Vulpian† had in fact just described the symptoms of disseminated sclerosis. Everywhere interest was taken in the strange manifestations of this affection, of which a learned diagnosis was gladly made in medical clinics.

An attentive study of Friedreich's cases made M. Charcot at first think that perhaps he was dealing with disseminated sclerosis having invaded principally the posterior columns.‡ Such a supposition must indeed have presented itself naturally to the mind of observers of that period, who had never seen cases analogous to those of Friedreich. Hence, M. Bourneville examines in a special chapter§ Friedreich's cases under the title, "Cases in which there exist simultaneously the symptoms and the lesions of locomotor ataxy and of disseminated sclerosis."

After having given the translation of two of the principal cases of Friedreich, M. Bourneville expresses himself as follows:—

"The mode of onset indicated is not that which is ordinarily seen in ataxy, whilst it is much more conformable to that of disseminated sclerosis. Difficulty of speech, trembling of the head, nystagmus, are morbid phenomena foreign to the classical type of ataxy. Finally, contrary to what takes place so frequently in this disease, cutaneous sensibility and vision were not affected."

And with regard to the second case:—"The onset here has occurred in the same way as in the first case. . . . Moreover, we see that the patient has had spasmodic contractions in the peronei muscles, stammering, nystagmus, enfeeblement of motor power and of vision, and moreover that cutaneous sensibility was intact.

* CHARCOT, Leçons sur les maladies du système nerveux, t. I (Clinical lecture of 1868). *See* Bourneville's digest in *Mouvement médical*, 1868, Nos. 197, 235, 259 et 327.

† VULPIAN, Note sur la sclérose en plaques de la moelle épinière. *Union médicale*, 1866.

‡ See FETZER'S "Klinische Vorträge von Charcot," Stuttgart, 1874, p. 521 quoted by Friedreich.

§ BOURNEVILLE, Nouvelle étude sur quelques points de la sclérose en plaques disséminées in BOURNEVILLE et L. GUÉRARD, De la sclérose en plaques, p. 191 Paris, 1869, chap. ii., p. 204.

"On the other hand, apart from lightning pains, there is nothing clearly belonging to ataxy, for the motor disturbances are so badly defined that one might ask if we are concerned with the trembling of disseminated sclerosis or with the inco-ordination of locomotor ataxy. . . ."

And M. Bourneville concludes:—"What is the outcome of this? It is that incontestably there is a mixture of two diseases . . . to wit, the co-existence in this case of progressive locomotor ataxy and of disseminated sclerosis."

Professor Charcot does not admit this combination of the elementary forms of these two diseases. "For myself," says he, in his Lectures on the Diseases of the Nervous System, "I have never met with, on the cadaver, the co-existence of multiple grey induration and posterior *fasciculated* sclerosis, and without denying that this association might exist, I believe it to be at least infinitely rare. It is pretty common, on the contrary, that the sclerous patches, which, as a rule, are principally seated in the antero-lateral columns, pass across the postero-lateral fissures, and encroach upon the posterior columns. Sometimes even I have seen them, having become continuous, occupy a large part of the thickness of these columns in the whole extent of one of the regions of the spinal cord—the lumbar, for example. Now, in all the cases of the latter kind, the ataxic symptoms had been, during life, manifested in different degrees."* And M. Bourneville adds in a note that the two cases of Friedreich, referred to above, belong to this category, what Charcot had not said expressly.

Friedreich [11] refutes this opinion by close reasoning, and demonstrates victoriously that his cases do not belong to disseminated sclerosis. "Why," says he, "has Bourneville made choice in my cases, reproducing some, and leaving others out? It is very surprising, for example, that he does not mention my first patient, Andreas Lotsch, since in this case the clinical and anatomical characters of degeneration of the posterior columns were certainly of the clearest." Friedreich then asks himself, by what signs one ought to

* CHARCOT, Leçons sur les maladies du Système nerveux, t. I, seconde edition. Paris, 1875, p. 243.

recognise "a true ataxic." Should it be, as Duchenne has laid down, by the absence of speech troubles? But one might upset this statement by arguing, on the other hand, that *true ataxics* are those in whom the want of co-ordination extends to speech and to ocular movements! "In my opinion," says Friedreich, "Charcot has attributed in my cases too great importance to nystagmus and to disorders of speech, whilst all the other more characteristic signs of disseminated sclerosis have been absolutely wanting during the many years that the patients have been under my observation. They have never presented diplopia, amblyopia, muscular contraction, spinal epilepsy, epileptic or apoplectic attacks, intellectual disturbance, bedsores, &c. The ataxic nature of the motor disturbances of my patients was so characteristic that it was impossible to doubt their significance, and because the ataxy invades the muscles of the tongue and those of the eyes, would that be a reason for not considering them true ataxics! Is it not, on the contrary, still more a reason for considering them as such?

"I find it incomprehensible," says Friedreich finally, "that anyone can still speak of disseminated sclerosis, when I have given the results of three carefully-made autopsies . . . and I hope that Charcot, in the vast field of observation which he commands, will sooner or later find a case analogous to those I have described."

This prediction was to be realised eight years later. I shall remark, moreover, that Charcot had never himself definitely pronounced on Friedreich's cases. He remained rather expectant, as we see by the quotations borrowed from his lectures. But in 1884, in one of his clinical lectures at the Salpètrière, he comes boldly from his scepticism, and shows a young patient affected with hereditary ataxy, which he for the first time considers as a special affection, clearly differentiated from disseminated sclerosis and from locomotor ataxy. One may say that from that moment Friedreich's disease was clinically recognised in official teaching; henceforth it will occupy a distinct place in nosography among spinal affections.

"This disease," says Charcot, "approaches in symptoms

at the same time, locomotor ataxy properly called and disseminated sclerosis, whilst it distinguishes itself from them entirely in certain directions; it has been called hereditary ataxy, or Friedreich's disease" (45).

Two years before had appeared at Montpellier the important work of Dr. Auguste Brousse, who was the first to propose the name of "Friedreich's disease," to designate the particular morbid affection so well described by the professor of Heidelberg. This work gives a complete *résumé* of the subject at that time. It appears interesting to reproduce from it the principal conclusions:—

"The disease studied by Friedreich," says Brousse (34), "under the name of hereditary ataxy, is a special disease having a proper etiology, symptomatology, and morbid anatomy. It is characterised—

"1. From the etiological point of view, by its development in childhood or adolescence, due to hereditary influence, direct or indirect; the two sexes appear equally predisposed.

"2. From the symptomatic point of view, by ataxy of the four limbs, commencing in the lower extremities and developing so as to reduce the patient to almost absolute functional impotence; by difficulty of speech, by absence or late appearance of sensory disturbances; by absence of trophic troubles and preservation of tone of the sphincters; by slow and continually advancing progress; by very long duration; finally, by an always fatal termination, generally brought about by an intercurrent disease.

"3. From the pathological point of view, by fasciculated sclerosis of the posterior columns of the cord, extending upwards to the bulb, complicated by diffuse sclerosis of the lateral and anterior columns.

"4. From the diagnostic point of view it must be distinguished from progressive locomotor ataxy, from disseminated sclerosis, and, lastly, from cases described in Germany by Westphal and Schultze under the name of combined degeneration of the columns of the cord.

"5. From the nosographic point of view it renders necessary the creation of a new class of chronic spinal affections—the class of mixed myelitis."

In England hereditary ataxy was first described before the Clinical Society of London, October 8th, 1880. At this meeting Dr. Gowers showed three patients belonging to a family of nine children, five of whom were ataxics. He specially insisted on the characters which absolutely distinguish this disease from ordinary locomotor ataxy. Gowers recalled on this occasion that Dr. Carpenter (6) of Croydon, in 1871, had brought before the same Society two sisters affected with a curious disease hitherto unknown, and which was no other than Friedreich's ataxy. Since then, a brother of these two patients has also been attacked by the same disease.

At the meeting of November 13th, 1871, at which Dr. Alfred Carpenter exhibited his patients, the Clinical Society appointed a committee, composed of Drs. Richardson, Hughlings-Jackson, Lockhart Clarke, and Broadbent, to examine these remarkable cases, and to report at the next meeting. These two sisters presented the important symptom of lateral spinal curvature already mentioned by Friedreich, and which has been recognised as one of the characteristic signs of hereditary ataxy.

In recent years, the number of cases of this disease observed in France, Germany, England, Italy and America has so far increased that M. Soca (91) in his excellent thesis, in which are recapitulated almost all the known cases, reckons up 165. But that is a very exaggerated total; for if the published cases are more closely examined and seriously criticised, it is soon perceived that the list contains a very great number of cases which are incomplete, doubtful, or even absolutely foreign to Friedreich's disease. Before proceeding to this critical review, which will allow us to eliminate many cases which are inappropriate, and which obscure the clinical picture, it appears necessary for us to fix first of all the essential characters of this picture. To this end I shall give first of all a *résumé* of a case of my own which has been under my care for three years (April 1886), and which is thoroughly typical of Friedreich's disease

A. K., aged twenty-four (in 1886), a member of a liberal profession, has been affected from the age of eight, and has been

unable to walk for the last three years, since he had acute articular rheumatism, convalescence from which was prolonged for several months, at the age of twenty-one.

Family History.—Father, aged fifty-six, is very robust, it is said, and has never been ill; I have no information as to his health or his habits. Mother in good health also, but very nervous.

On the paternal side are several uncles and aunts, it appears, who are more or less affected with nervousness and peculiarities of character. There are some "hot-headed" members in the family. But no one is known to have presented symptoms analagous to those of our patient. A younger brother, aged sixteen, only walked when three-and-a-half years old; he is imperfectly developed for his age, very nervous, and of an irritable and troublesome temperament. A younger sister is in good health, but is also nervous.

Patient's History.—His early childhood presented nothing particular. Suckled by his mother, the child developed normally, and walked at fifteen months. Very intelligent and precocious for his age, he early learned to read and write. At the age of eight the first difficulty in walking appeared, a certain weakness of the legs and of the movements, which was looked upon as "nervous." He frequently fell; he walked slowly, and he planted his feet one before the other. He was scolded, because it was looked upon as carelessness and idleness. Nevertheless, he was able to walk easily alone. He never complained of pain. About two years after, he became ungainly with his hands, and five years only after the onset (the first symptoms in the legs) his speech began gradually to become drawling and difficult. The boy was then aged thirteen. Never involuntary grimaces, but so-called nervous movements in the arms and legs during walking. One day when the child was suffering from headache and stomach ache, a medical man was called in, and his visit was taken advantage of to consult him on the singular movements observed in the little patient. The doctor had him undressed, made him walk before him, examined him carefully, and said: "Oh, it will be nothing, it is St. Vitus's dance"—ordinary chorea slightly developed. Now this supposed St. Vitus's dance has never disappeared, on the contrary, it has been accompanied little by little by other symptoms, so that the disease has insensibly become aggravated. For three consecutive years, however, the patient was treated for St. Vitus's dance.

At the age of thirteen, the boy, who was well-advanced at school, was apprenticed to a lithographer, with whom he remained

three years without making any progress. The employer said that he was too weak to follow this trade, and that he would never make anything at it. We must suppose that the ataxy of the arms, which had become more and more developed, was an invincible obstacle to the work of the young apprentice. He was then sent to college, and at the end of some months he had already outstripped his companions by the vigour of his memory and intellect. Henceforth he advanced rapidly and did not cease to devote himself to intellectual labour. However, his gait became more difficult, his handwriting more slow and trembling. At the same time his speech also became slower. At the age of seventeen the patient began to make use of a stick for walking, and henceforth did not give it up. But the progress of the disease had been so insensible and so slow for several years, that he attached scarcely any importance to these symptoms, and the less so as they were not at all painful. He did not perceive, so to speak, that his disease was progressive. Thus he reached the age of twenty-one. At this period he was attacked with acute articular rheumatism, which confined him to bed for three months, and at the end of several months' convalescence he perceived that he could no longer walk alone. In darkness especially he felt less firm on his legs, and for a long time, even before his rheumatic attack, he could not go out in the evening, whilst during the day he still walked pretty well with the assistance of a stick. His legs had become more and more weakened, and even while giving one arm to someone, and helping himself with a stick with the right arm, the patient continued to find more difficulty in walking.

Never has he experienced lightning pains, nor girdle pains. Never incontinence of urine, nor any other urinary trouble. No disturbance of vision. Habitual constipation. No seminal losses. Never syphilitic infection.

Present Condition.—(He was examined several times from April, 1886 to April, 1889). Body generally imperfectly developed. Muscular system rather feeble. Adipose tissue scanty. General pallor of integuments. Skeleton fairly formed. Head large, round, symmetrical, brachycephalic. Scoliosis, with convexity to the right very pronounced in the region of the dorsal vertebræ, the ribs bulging on the same side forming a well-marked prominence under the scapula. No other peculiarity to note as to the development of the skeleton except the deformity of the feet which we are about to describe, remarking that it is not of bony nature.

The feet are small, deformed, affected with talipes equinus,

and as if shortened. Their colour is of a darkish blue. They feel cold to the touch. The sole of the foot is much hollowed, and has undergone a certain twisting, which is seen by the transverse furrows of the skin, which have become oblique from within out, and from behind forward, as if the anterior part of the foot had been twisted on the posterior. There is no permanent contraction of the sole of the foot. The dorsum of the foot is prominent; there is a projection at the level of the tarsus. The toes, especially the great toe, are raised up clawlike, and the tendon of the extensor proprius pollicis appears prominently under the skin. These symptoms are especially marked when the foot is raised; they are largely effaced when the patient places his foot on the ground. The articulations are flexible. All passive movements are easily performed. The tibio-tarsal articulation is relaxed, and voluntary extension of the foot can be prevented by fixing it with the hand. During walking the toes are constantly affected with movements of athetosis.

Motor symptoms.—The patient cannot stand upright without support. His trunk, head, and limbs are always in motion. He says that he could never be photographed because he is too nervous, and his features are too mobile. In reality it is because he cannot hold still an instant; even when he is at rest, his head and his trunk are incessantly agitated. It is static ataxy, as Friedreich calls it; when seated in an armchair he is perpetually moving. They are a kind of badly marked choreiform movements. We can understand that at the commencement of the affection the disease was mistaken for chorea. We shall speak of it again in treating of the diagnosis. Let us add, however, that this "choreiform instability," as Charcot calls it, is not always alike. Sometimes very pronounced, it is much less so on certain days, especially if the patient is well rested.

All voluntary movements of the arms and legs can be executed, but are manifestly ataxic. It is impossible for the patient, when told, to touch with his index finger, at the first attempt, the tip of his nose or the lobule of his ear, even with the eyes open. He always places his finger at a certain distance from the point intended, then, without leaving the surface of the skin, he makes it glide slowly to the indicated spot. The patient, whose intelligence is highly developed, is well aware of how his actions proceed. At the moment of raising his index finger to execute the required movement, he knows very well where he is going, but he soon loses control over the direction of his arm, because he has no longer an exact notion of the position of the limb during move-

ment. We see it then perform oscillations which distantly recall the trembling of disseminated sclerosis, but do not sensibly increase in intensity at the final moment. If the patient is told to take up a small object from the table—a pencil, for example, a match, or better still, a pin—he advances his hand above the object on which his eyes are fixed, makes it hover an instant, hesitates, separates somewhat his fingers, then darts suddenly on the object to seize it as a bird of prey would do, with this difference, however, that the direction of the movement is sinuous and interrupted, instead of being direct as in the case of the bird who arrives by a straight line on the coveted object.*

The method of picking up the pin and the pencil from the table is also characteristic. The patient employs to this end all his fingers, which he keeps separate and then brings together, making the object glide into his hand till he holds it conveniently.

He buttons his clothes with difficulty, especially the small buttons. Nevertheless, even if his eyes are closed, he ends by reaching the small buttons, and when once they are held, he does not relax his hold. He cannot dress or undress without assistance, on account of the effort required to lift his clothes. At the moment of taking off his shirt, when it passes above his head and covers his eyes, he loses his equilibrium completely on the stool on which he is seated, falls backwards and remains with his legs in the air till someone comes to his assistance. However, he has made in the meantime great efforts to regain his equilibrium.

It is the incessant movements of the trunk and limbs which prevent the patient maintaining the upright position. If he is told to loose hold of his support, and he sees around him those who are ready to support him, he can remain standing an instant, his legs widely separated, and even if his eyes are closed he is not more unsteady at first; but his unstable equilibrium is soon disturbed, a more extensive movement of the body makes him lose his balance completely, and he would collapse if not supported immediately (Romberg's symptom).

In spite of this, however, it is easy to assure oneself of the integrity of the muscular sense. The patient distinguishes very readily, with his eyes closed, different coins placed in his hand, in the right hand equally with the left. He estimates also pretty correctly the difference of weights when he supports small objects in the hand.

* Carre had already observed this movement of the hand, which "hovers" over the object before seizing it, but it is to M. Charcot that we owe the comparison with the bird of prey, which appears to me appropriate and striking.

Dynamometer: right hand forty-one, left thirty.

The patient feeds himself but slowly, and with some difficulty.

The patient writes very well, although rather slowly. The letters are well formed, and not at all shaky. The writing is plain and easily read.

The muscular strength of the arms and legs is well preserved. His leg cannot be flexed with both hands when he maintains it in extension. There is marked enfeeblement of the movements of the feet. Since the patient has been unable to walk alone he thinks he has noticed certain changes in his gait. Formerly he walked more on the soles, the fore part of the foot; now more on the heels, after the manner of ataxics. At present he cannot make a single step without being supported on each side. When he wishes to walk he passes his left arm under the right arm of the person who strongly supports him, and he supports himself with a stick in the right hand. He advances then slowly and with difficulty, making each instant extensive movements of the head and trunk, whilst the inco-ordination of movement of the right arm, which holds the cane, causes the latter to go from side to side so that it often misses the ground, and makes the patient plunge forward at the moment that he felt secure of finding a support.

He drags his feet in walking and controls their movements by sight; at each step the point of the foot deviates inwards, and rubs on the ground, especially the left foot, whilst the heel scarcely touches it. The leg, moreover, is not thrown strongly forward as in ordinary ataxy, for the foot scarcely leaves the ground, and the gait strongly recalls that observed in spastic paralysis, except that the stiffness of the legs does not exist here. The steps are short, unequal and irregular, and when one foot has been put forward the other follows, its fore part being dragged along the ground, deviating strongly into equino-varus, whilst the trunk and the head are thrown back in a disorderly manner. When the foot is raised from the ground the inco-ordination of movement becomes very manifest; the leg is flung forward, and the heel falls heavily. To sum up, what characterises the gait is the fact that the trembling is more pronounced than the ataxy. With the least emotional disturbance, the patient can no longer walk. His legs give way under him. If he is tired his feet continually turn over, and he catches against every stone in the way. He can with difficulty mount stairs, but he descends them with yet greater difficulty.

When I took his foot-prints, after the method of M. Gilles de la Tourette, it was necessary to support him under the arms to

make him walk on the band of paper. This is what explains the absence of the characters which we have just described—the dragging of the front of the foot, and the heel hardly touching the ground. When supported the patient raised his feet, threw forward his legs and brought down the heel at every step. What the print especially verifies is the deep concavity of the sole of the foot, which is shortened, and the trembling, irregular walk of the patient, whose legs double up several times under him during the experiment; lastly, in several prints we find the double heel of ataxics.

The sensibility of the soles was, moreover, so exaggerated that the floor caused an insupportable sensation of cold, and the pressure caused pain; he cried out while taking the first steps with bare feet. The toes were seen to be raised violently in a spasmodic way and the sole to become more hollow each time that the patient raised the foot. The feet very quickly became cold and more and more cyanosed. It was then very difficult to warm them. "When my feet become warm again," said the patient, "they readily perspire, and quickly lose their heat." He adds that the impression of cold to the feet was very painful to him and paralysed him. He felt plainly the ground and its inequalities, and did not experience any peculiar sensation in the sole of the feet as is the case in tabetics.

Disturbances of Speech.—It was towards the age of thirteen years that difficulty of speech was noticed. His parents remarked that the boy's words became drawling and thick. He did not himself notice it—at least he cannot fix the time when speech troubles first manifested themselves. He does not recall that he may have had an interval between the symptoms in the legs and arms and those of speech. He maintains that they have all been developed almost simultaneously.

His voice is monotonous, very low-pitched. Speech is slow, somewhat scanned, but without hesitancy. The mechanical articulation of the sounds and the duration of the words are alone affected. There is no stammering.

The patient pronounces long words with a single effort, although slowly, and somewhat scanning the syllables; but he has no hesitancy of speech, does not cut the words in two, does not stammer. He will say without stopping, but slowly, i-na-mo-vi-bi-li-té, all in the same drawling tone, no syllables being pronounced more rapidly than others. The small pauses are equal between each syllable. Whilst the general paralytic slurs his syllables, this patient articulates distinctly in a slow, monotonous, and drawling voice.

The tongue, when protruded, is of normal size. But it undergoes very marked ataxic movements; it contracts, it is suddenly drawn to one side, then is withdrawn, curving up behind the dental arch, to come out again with a sudden jerk. His reading is also slow and slurred. The soft palate reflex is absolutely normal. The knee reflexes are completely abolished.

Nystagmus.—Horizontal oscillation of the eyes when following a moving object is the last symptom which we should connect with ataxy. Nystagmus is not very pronounced. It must be looked for and brought out to be observed. It is not present when the eyes are at rest. It is a motor ataxy of the ocular muscles, a dynamic nystagmus, in opposition to a static nystagmus, a balancing of the ocular muscles in different directions, which is observed when the eyes are fixed on a point. The patient says that when he fixes an object his sight is soon fatigued. The oscillations of nystagmus are very rapid and of small amplitude; they do not exceed a millimetre in extent, and only take place at the moment when the eyeballs reach the corners of the eyes. However, they are still observed, though more rarely, when the eyes are directed in front; at the moment when the eyes are fixed on the finger placed a few centimetres before the forehead, opposite the root of the nose, some slight oscillations are seen to occur. It is what Friedreich had already clearly observed, remarking that in ordinary cases the existing nystagmus is thus made to cease, instead of being provoked.

Impulse to laugh.—The patient is often taken with an impulsive desire to laugh. He notices carefully these occurrences, which come on him unawares, and which he cannot resist. Lips habitually separate.

Sensibility absolutely normal in all its manifestations, to contact, pain, temperature and electricity. Nowhere in the legs are anæsthetic patches. He feels everywhere the lightest touch, and localises pin pricks perfectly. Never creeping sensations nor pain in the legs, particularly lightning pains. When the patient walked much, he experienced for several months a pain in the left knee. This articulation has never been swollen. Passive movements have always been free; the knees, left as well as right, supple and flexible. He then experienced great weakness in the legs, following fatigue; he could hardly drag himself along. He felt as if his legs were too long, "the nerves unstrung," especially during the north winds. The pain in the left knee then sensibly increased. He could no longer take long steps as usual. His feet dragged on the ground. This is why the patient

said: "The smaller the steps, the more awkwardly I walk." The pain, which he attributed to a rheumatic origin, has completely disappeared since the patient has been less on his legs, and has scarcely walked at all.

Pupillary reflexes perfectly normal to light, to convergence, and to accommodation. No diplopia nor strabismus; sight excellent.

Sphincters normal. The patient is not subject to constipation. He can retain his urine for long. No gastric troubles.

Genital functions.—No disturbance to note. No involuntary emissions. Erections normal. No impotence nor premature ejaculation.

Psychical functions normal. Intelligence remarkable, especially the poetic and imaginative faculties.

Electric reactions normal everywhere except in the muscles of the leg (antero-external group), where electro-muscular contractility is weakened.

During electric examination the patient, seated on a stool, cannot be left alone an instant; he loses his equilibrium at every movement, and as he is perpetually in motion he would fall if not watched. (Choreiform instability, static ataxy.) Moreover, he is weighed down, he is doubled up, and his head falls on his breast.

Galvanic Exploration of Nerves.—L. ulnar (K.C.C.) 1·5 ma.; R. 1·5. L. radial, 3; R. 3. L. peroneal, 2; R. 3.

Faradic Exploration of Muscles.—Tibialis anticus, R. 75 mm.; L. 80. Peroneal muscles, R. 85; L. 84. Gastrocnemius, R. 78; L. 82. Vastus ext., R. 83; L. 80. Interossei, R. 76; L. 80.

Galvanic Exploration of Muscles.—Tibialis ant., R. 8·5 ma.; L. 8. K.C.C.=A.C.C.; occasionally A.C.C.>K.C.C. Peroneal muscles, R. 7; L. 7, weak. Contractions normal, rapid. No R.D. Legs thin; atrophic on antero-external aspect.

Taking for our basis this case, and others which we have had the opportunity of examining in the wards of M. Charcot at the Salpêtrière, and of M. Dejerine at Bicêtre, and comparing them with the cases that have been hitherto published, we shall successively consider Friedreich's disease from the etiological, the symptomatic and the diagnostic point of view. Finally, we shall describe its pathological lesions, which class this affection among the combined scleroses of the spinal cord.

ETIOLOGY.

This case is distinguished from most of those which have been previously observed, by the absence of the *family* or *hereditary* (generic, as Smith calls it) character, which has for long been considered as the rule in this disease. Indeed, the brother and sister of the patient have not been attacked by the same affection, and if we credit M. Soca, who has deduced the law, from the observations of several authors, among others Gowers ([25]) and Rütimeyer ([41]), that the children of the same family become affected almost at the same age, we should be able to say that the patient's brother and sister no longer run any risk of having this affection, since they have both passed the age at which the disease commenced in their brother.

M. Soca formulates this law in the following way:— "Friedreich's disease commences at the same age, within two or three years, in all the members of the family which it attacks."

We shall, however, make some reservations on this point, and before adopting this law, the practical importance of which would be very great, we think that it is expedient to wait for a greater number of cases, and especially greater exactness among authors in fixing the onset of the first symptoms. Mr. Soca says so himself (p. 132):—

"The first phenomena which strike the patient and his friends vary somewhat in different cases, sometimes the onset of the disease dates even from birth. The little patients never learn to walk properly. It is only very late that they walk alone, and then their gait already presents all the characters of ataxy. Let me add that this mode of onset is pretty common."

In reality, the onset of Friedreich's disease is so often so insidious and insensible that the patient and his friends do not trouble about them till long after their appearance, when the weakness of the legs and the ataxy of movement have become sufficiently developed to interfere with walking. If the physician who is at length consulted then questions the parents they will tell him that they have noticed, for several years perhaps, certain signs which should have aroused their

suspicions, but without giving serious attention to them, because they attributed them to idleness and obstinacy on the child's part rather than to disease.

Must we then admit that external conditions, environment, occasional causes, are really without any influence on this disease which would thus fatally attack at the same age the members of a family? We must confess that our knowledge of the etiology of this disease is still so obscure that it is actually impossible to say anything on the subject. We think, however, that Friedreich's disease is no exception to the general laws of heredity, and we believe that one would commit a great error in wishing to withdraw it from external influences (always necessary for the development of hereditary morbid germs slumbering in the organism) by reason of the absolute ignorance in which we are of the occasional causes of this disease.

It has been observed in several attacks that the onset of the disease has declared itself after an acute disease—a fall, an injury, or after early habits of onanism. These are the common causes of which we must assuredly take account, but which can only be of influence in individuals hereditarily predisposed.

In the personal history of patients we sometimes find noted scrofula, tuberculosis, and infantile convulsions. But it is the eruptive fevers and typhoid fever which appear to have most often been the occasion of the onset of the weakness of the legs and the ataxy of the extremities. In one of Rütimeyer's patients the first symptoms of ataxy were manifested during a long convalescence following scarlatina of four weeks' duration. Vizioli ([59]) has seen two cases of Friedreich's disease develop after scarlatina. Ormerod ([54]) also quotes a similar case. Whooping-cough and measles have also been observed to have preceded, by a longer or shorter period, the first manifestions of the disease. Rütimeyer and Vizioli have each seen a case in which the ataxy of Friedreich appeared after typhoid fever. Let us further note as antecedents the mention of febrile affections not attended by eruptive or cerebral manifestations.

Musso ([49]) insists at some length on small-pox as a

predisposing cause of the disease. He quotes numerous examples of affections of the nervous system observed after small-pox by Gubler, Westphal, Leyden, Bernhardt, Seppilli, &c., and thinks that in three of his patients it is the small-pox which has kindled the disease, which would otherwise perhaps have remained latent all through life. But in examining Musso's case more closely, it may justly be asked if the first symptoms of ataxy should really be attributed to small-pox. Indeed, in one case, the ataxy did not appear till two years after, in another two years and a half, and in the last not till three years after a mild small-pox.

It is to be remembered that our patient experienced a notable aggravation of his disease after an attack of acute articular rheumatism. At the end of convalescence from that disease he could no longer walk alone, as he could previously with the aid of a stick. In Friedreich's seventh case there is an analogous circumstance. Louise Schulz, whose disease commenced at the age of thirteen, was attacked at fourteen with acute articular rheumatism, after which the shaking of the limbs greatly increased, and speech became indistinct and stammering, which was not the case previously.

In other patients chorea has been mentioned as a pathological antecedent. Thus Rütimeyer ([41]) says that Jacob Blattner (his seventh case) had at the age of four years St. Vitus's dance for six months, and that after that affection he remained feeble in his legs and uncertain in his gait; since then, too, the disease invaded successively the upper extremities and speech, as in his eldest sister. We ask ourselves if this chorea should not rather be considered as a symptom of the onset of Friedreich's disease. We have seen that our patient was regarded for three years as suffering from chorea, and we know now that "choreiform movements" form part of the regular procession of symptoms of this disease. We think that chorea should henceforth be considered as belonging to the first period of Friedreich's disease, whenever the latter will appear to be the direct consequence of it. Other concomitant symptoms, such as the abolition of the knee reflexes, will serve in a doubtful case to settle the diagnosis.

Let us relate yet a negative etiological fact, which assumes a certain importance in the enumeration of the causes of so-called hereditary ataxy. We hardly ever find syphilis mentioned in the personal or hereditary antecedents of Friedreich's ataxics—contrary to what is observed so frequently in true tabetics.

On the other hand, alcoholic excess in the parents has been pretty often blamed. Friedreich was the first to insist on this point. The first six cases that he observed belonged to two families, Lotsch and Suss. Now, the fathers of these two families were drunkards, and the widow of Suss said that all her children were conceived during drunkenness. Other observers, notably Everett Smith, Quincke* and Rütimeyer have since related analogous examples. There are other cases, as a set-off, where alcoholic excess of the parents is absolulely excluded.

Direct heredity has, therefore, some share in the symtomatology of Friedreich's disease, as in that of myopathic atrophy and Thomsen's disease. There are, however, numerous exceptions to this rule, and isolated cases like the present one are to-day not rare in science. It would not be necessary to think on this account that these last cases escape the law of heredity. We establish most often, as in our patient, amongst ancestors and relatives nervous and psychical affections of diverse nature, so that these sporadic cases fall like the others under the general law of neuropathic heredity, although they do not present the "family" character, which is the index of an accumulated converging hereditary influence, that is to say, raised to higher power. (See subjoined the genealogical tables of Rütimeyer, Musso, Vizioli and Gowers.)

Rütimeyer (*Virchow's Archives*, 1883, vol. 91, p. 116 and 117). Blattner "Stulzi" (that is to say, "hobbling") married in 1710.

All the direct descendants of this Blattner are entered under this surname "Stülzi," in the official registers of the commune of Küttingen up till the "forties" of the present century, and the strange gait of this man must have strongly impressed his con-

* Quincke, in Friedreich's second paper, *Virchow's Archiv*, Bd. lxviii, p. 165, 1876.

temporaries for it to have remained during so many generations in the recollection of the inhabitants of the country. However, the eight cases of Freidreich's disease have only quite recently shown themselves in this Blattner family, whilst previously neither nervous nor mental diseases were known in it.

These eight cases belong to four different branches, of which the great grandfathers were brothers and lived at the commencement of the present century, direct descendants of the famous Blattner-Stülzi. Here are the four branches (in the fourth generation probably, and not in the second, as Soca erroneously states).

BLATTNER-BLATTNER.
Ten children, of whom seven healthy.

Rudolf (1). Born 1865. ATAXIC (scarlatinal).
Gottlieb. Born 1848. ATAXIC.
Marie. Born 1853. ATAXIC.

BLATTNER-BASLER.
Seven children, six healthy.

Karl. ATAXIC. Mother affected with chorea in childhood. Maternal uncle paralysed in both legs suddenly (acute poliomyelitis).

BLATTNER-KYBURZ.
Seven children, six healthy.

RUDOLF (2). Born 1867. ATAXIC (following typhus).

BLATTNER-WEHRLI.
Ten children, seven healthy.

Marie Madeleine, age 20. ATAXIC.
Jacob. Died, aged 18. ATAXIC.
Fritz. Died of measles, aged 9. ATAXIC.

As for age and sex, we have but little to say of them. Friedreich thought that hereditary ataxy was a disease commencing at puberty, especially in girls. This observation is not always verified. Many cases date from infancy as well in boys as in girls. After the age of twenty it is extremely rare for Friedreich's disease to show itself.

Symptomatology.

The clinical history of our case furnishes a typical picture of the symptomatology of Friedreich's disease, which may thus be recapitulated: slow and progressive ataxy of the four limbs—usually attacking several children of the same family, dating often from very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and of the eyes; weakness of legs, increasingly difficult gait (tabeto-cerebellar, Charcot); choreiform unsteadiness; static ataxy (Friedreich); difficulty of articulation; nystagmus; spinal curvature; paralytic club-foot; abolition of knee-reflexes; no sensory disturbances; absence of oculo-pupillary anomalies and of lightning pains; integrity of sphincters.

Sometimes other symptoms are added which are due to

complications, or which result from an error of diagnosis. It appears to us necessary from this point of view to eliminate a certain number of cases which are still classed under Friedreich's disease, although they have no right to be so considered. In reckoning these doubtful cases the symptomatology of this affection is confused, and elements of error are introduced which obscure the clinical picture of it.

Let us rapidly pass in review some of the cases which in our opinion ought no longer to figure in the category of Friedreich's ataxics.

There are first of all three patients of Hitzig,* referred to by authors and hawked about from one bibliography to another without any verification of the reference. As M. Soca remarks, this reference is inexact. It refers in fact to page 513 of the *Berliner Klinische Wochenschrift* of 1875,which is devoted to advertisements! In reality, it does not relate to a work of Professor Hitzig on Friedreich's disease, but simply to a discusion which took place at the Berlin Society of Medicine on January 20th, 1875, after a communication by Dr. Bernhardt on muscular atrophy. Hitzig said that he had had under observation for several months two brothers and a sister affected with muscular atrophy, in whom the disease had existed between five and ten years; seven other children of the same family perfectly healthy. The *mother* of these ten children was *tabetic* in a high degree. But there is not a word about difficulty of walking, which should have been observed in the little patients.

The error proceeds from Seeligmüller,† who thought that the atrophics of Hitzig ought to be classed under Friedreich's disease, perhaps on account of the tabes of the mother, and who speaks of difficulty of walking (*Gehstörunsgen*, loc. cit. p. 188), whereas there is no mention of it in the report of the discussion.

Seeligmüller, moreover, is not fortunate in the choice of observations on which he relies for the description of Fried-

* *Berliner klinische Wochenschrift*, 1875, *p.* 143.

† V. SEELIGMULLER in Halle, Krankheiten des Nervensystems II. Scleroses der Hinterstränge, p. 185.—In GERHARDT'S *Handbuch der Kinderkrankheiten*, vol. v., part ii., first edition, Tubingen, 1880.

reich's disease. Beyond the cases of Friedreich—the only really authentic ones at that period—he quotes *Du Castel*,* whose patient was not ataxic but paralysed; the two cases of Kellog (⁸), too incompletely described and presenting too many anomalies to be admitted as belonging to Friedreich's ataxy; the cases of *Bouchut*† (in which ataxy exists in name only), a boy eleven years of age, ill for fifteen days with tingling in the feet, sometimes diplopia with convergent strabismus and slight constipation, and a girl of fourteen years, an old rachitic subject, ill for two years, who walks with a firm but slow step, without trembling and without defect of muscular co-ordination; moreover, "one may tickle in any way the soles of her feet without making them move" (!!); lastly, the case of *Kahler* and *Pick* (¹⁵), combined sclerosis of the posterior and lateral columns, which is by no means a case of Friedreich's ataxy, the dominant symptom being paraplegia.

Let us add, that to crown his mistake, Seeligmüller (²³) has published two cases which certainly do not belong to Friedreich's disease; that Friedreich himself, consulted by the author, has refused to recognise them as analogous to his own, but which Seeligmüller persists in spite of Friedreich's authority, and in spite of evidence, in considering as authentic examples of hereditary ataxy with nystagmus. These are the cases:—

Leo von K——, age twenty-six, descended from an old noble family, in the genealogic tree of which neuro-psychopathies abound. Uncertainty of gait at twelve years, at fourteen progressive myopia, from fifteen to sixteen melancholia. Onanism. Sexual perversion. At twenty-one he enlists as a volunteer in the cavalry. He is a bad horseman. He has learned to swim, but he is a bad swimmer. He dances, but he is a bad dancer, for he easily becomes giddy if he turns round in dancing, and if he rides round. Girdle pains. Urinary troubles. Various psychical disorders. What is most striking in his physiognomy is his nystagmus, which the patient can arrest when he wishes,

* Du Castel, Observation de sclérose primitive des cordons de Goll. *Gazette médicale de Paris*, Jan. 17, 1874, No. 3, p. 33.

† Bouchut, Ataxie locomotrice et sclérose des cordons postérieurs de la moelle chez les enfants. Signes ophthalmoscopiques. *Gazette des Hôpitaux*, March 31, 1874, No. 38, p. 297.

so much control has he over himself! He is able to arrest the nystagmus when he fixes his look fifty centimetres in front of him. He then feels firm on his feet. The patient is never at rest, but he can always arrest his movements by an effort of will. No locomotor ataxy. With eyes closed he goes pretty straight to a fixed point in front, but his legs are too wide apart and he walks irresolutely. He mounts a chair, &c., without difficulty. Plantar reflex abolished; cremasteric absent on the right side. *Patellar reflexes very exaggerated.*

Alexander von R——, age twenty-eight, married, robust, very strong, but awkward. From childhood a grimacer; shakes his head; spasms; rabbit-like movements of the nose and lips. Syphilis. Gonorrhœa; vesical catarrh followed by violent pains in the right hip. For six months he has had difficulty in walking (October, 1878), yet he has walked for several hours—limping rather—to go to the chase. Asymmetry of face. Left strabismus. No nystagmus. Speaks very quickly, and gets flurried. Ataxy of thought, but no trace of ataxy of speech. No spinal curvature. Walks with his legs apart, and with swaying of the body. His eldest daughter has the same gait. When upright, with eyes open, the patient does not stumble, nor does he if his eyes are closed. No trace of ataxy in the upper limbs. Plantar and cremasteric reflexes abolished. Knee reflexes very lively.

A sister, whom Seeligmüller was not allowed to examine, suffered, it appears, from symptoms in all respects like those of her elder brothers.

It is indeed sufficient to enumerate these symptoms to understand that Friedreich had good reason not to find in them an image of the disease which he had so conscientiously studied and described. Nor is Seeligmüller's indignant exclamation in any way justifiable, when he says (*loc. cit.*, p. 242): "To conclude, I must protest strongly against the assumption of Bourneville, who argues that the hereditary ataxy of Friedreich is a disseminated sclerosis, with predominant lesion of the posterior columns. The clinical picture offered by the patients of Friedreich, two of whose ataxics I have myself seen at Heidelberg in the autumn of 1878, as by my own is absolutely different from that of disseminated sclerosis."

Who would have doubted that Seeligmüller had seen Friedreich's patients? Doubtless Bourneville was deceived,

but we must confess that nothing resembles more a patient affected with disseminated sclerosis than an ataxic of Friedreich, with his choreiform unsteadiness, his nystagmus and his slow and scanned speech. It seems quite natural that one may have supposed, in 1869, at the time when disseminated sclerosis had just been discovered, and was not yet known under all its clinical varieties, that the first cases of Friedreich might be classed under that disease. We have already seen how Friedreich has replied to it; we shall not return to it.

Hammond ([28]) has enriched the statistics of hereditary ataxy by twelve cases, of which the very incomplete, superficial and aphoristic observations do not permit us in the case of any of them to subscribe without reserve to the diagnosis. It is absolutely certain, on the other hand, that most of these cases do not belong to Friedreich's disease. What can the following cases, for example, have to do with this affection? In the family of R—— there are three brothers who died young, and a sister in good health, of whose heredity Hammond says nothing.

1. A little boy, G. R——, ill since dentition, general paralysis and articulatory trouble; no pains; sensibility always normal; died aged five years.

2. His brother, L. R——, paralysed at six months; paralysis gradually increasing; died aged three years.

3. A third brother, W. R——, died aged three years; paralysis and articulatory difficulty, without atrophy and anæsthesia.

And authors would regard these patients as cases of Friedreich's ataxy! The twelve cases of Hammond should henceforth be erased from the list of this disease.

As much may be said for the two cases of Drs. *Oxley* and *Pollard* observed at the Liverpool Children's Hospital, and reported by *Davidson* ([31]).

Case 1. (Oxley).—M. B——, girl, aged nine years. Six weeks ago she was attacked without known cause, in two or three days, with muscular inco-ordination of the four limbs, as if she had chorea. Mastication and deglutition difficult. Speech embarrassed. Urine in the bed. Patellar reflexes exaggerated. Sensibility normal. Rapid improvement by appropriate hygiene and good nourishment. After five to six weeks she could walk alone.

Case 2. (Pollard).—J. G——, little boy, aged seven years. Ten weeks previously an attack of rheumatic fever; for the last eight weeks has entirely lost speech and the use of his legs. At rest in bed no involuntary movements. As soon as he wishes to make a movement, the latter is exaggerated and violent as in chorea. For a reply he makes a motion with his head. Sphincters normal. No exaggeration of tendon reflexes. Sensibility normal. Six weeks after entering the hospital he could walk alone, and stand with his eyes closed. Speech unimpaired.

It must be said that Dr. Pollard does not consider this patient as an ataxic of Friedreich, but considers him to be affected with paresis and functional ataxy, recalling certain hysterical manifestations.

The custom which has been followed, since Seeligmüller, of considering exaggerated or simply normal knee reflexes as belonging to the symptomatology of Friedreich's tabes, has contributed largely to lead astray the authors who have considered under the heading of this disease a number of cases with exaggeration of the knee reflexes, which ought not to figure in it.

Seeligmüller recognises that the symptoms of his patients differ from those which had been given by Friedreich as characteristic of his disease. In his manual he mentions in passing hereditary ataxy in the middle of the description of the symptomatic forms of tabes, and says ([85]): "The symptoms of the cases observed by other authors and by myself offer a clinical picture more or less different" (*in mehrweniger ausgesprochener Weise !*) to those of Friedreich.

I shall yet quote the two patients of *Massalongo* ([48]), the one of Descroizilles ([68]), and the one of Dalché,* which certainly do not belong to Friedreich's ataxy. The abolition of patellar reflexes is an absolute rule in this disease, and one will do right always to mistrust the diagnosis of this disease whenever the knee reflexes will not be lost, although all the other symptoms would seem to confirm it.

Another category of patients, on the other hand, in whom the tendon reflexes are abolished, have also been improperly reckoned among the cases of Friedreich's disease, and should

* P. Dalché, Maladie de Friedreich. Pseudo-tabes. Discussion. *Progrès médical*, June 30, 1888, p. 507.

no longer be classed under it. The anomalies which they present suffice to remove them from this affection, and to place them either under disseminated sclerosis, under the combined scleroses, or amongst other spinal or general nervous diseases.

Brousse's patient, for example (34), whose first symptoms, weakness and inco-ordination of the legs appeared only at the age of twenty-four years, had at the end of his life apoplectic attacks which do not form any part of Friedreich's disease. Moreover, the ataxy and the articulatory troubles have peculiar characters foreign to this affection. This is how M. Brousse expresses himself:

"Walking is not possible without assistance. When it is attempted it causes trembling of the whole body. . . .

"When the patient is at rest, if he is questioned rather suddenly his head is affected with trembling. . . . There is difficulty in his speech, it is hesitating, stammering."

The autopsy revealed a combined sclerosis, fasciculated of the posterior columns, and diffuse of the antero-lateral, with central myelitis. It was not therefore Friedreich's disease.

The cases of *Erlenmeyer* (10), *Hollis* (26), *D'Arcy Power* (32), *Fazio* (56), and many others besides, are doubtless liable to the same objections. The case of Carre (7), too, offers certain symptoms which render it suspicious. The disease began at twenty-two years by pains in the sole of the feet and in the thigh, then on the inner side of the hands, which were *dead* (sensibility blunted). Diplopia and weakness of sight. Speech impeded; a kind of stammering; one would say that the tongue, hindered by an obstacle, has difficulty in detaching itself from the palate, *then a series of words succeed one another rapidly*—all that is absolutely foreign to the symptomatology of Friedreich's disease.*

* I had written this critical review of cases of Friedreich's disease before reading the excellent article which Professor F. Raymond has devoted to tabes dorsalis in the Dictionary of Dechambre (See Bibl., No. 62). I see with pleasure that M. Raymond no longer admits the following cases as examples of the disease described by Friedreich—(1) the case of Kahler and Pick (35); (2) the two cases of Seeligmüller reported above; (3) the case of Brousse; (4) Hammond's case. The author remarks, moreover, that Carre's

After throwing out many cases considered by different authors as belonging to Friedreich's disease, we propose, as a set off, to add one which appears to us truly typical. We found it in the work of Topinard (4), and as it has passed entirely unnoticed, we give of it the following *résumé:*

Case 215.—Edouard L——, aged twenty-one years, tiler. Father and mother in good health. Eight brothers and sisters, of whom five have died before the tenth year. Scrofula; onanism from the age of thirteen, afterwards venereal excess. Periodic migraine every fortnight. Towards the age of eleven years, at la Pitié, M. Michon proposed an operation to remedy a sinking of the left scapula. Towards the age of seventeen, vertigo and cramps. His legs supported him with difficulty; tendency to fall backwards. His speech became difficult. He became humpbacked. Blows with a stick on the back, followed by dull pains for a month. To sum up, during two or three years the above-described phenomena; failures of equilibration, difficulty of pronunciation and gradual deviation of the trunk constituted the whole disease and became more and more pronounced.

Intellectual power excellent; sight and hearing excellent. Neither strabismus nor diplopia. Since the invasion of his disease, no headache, loss of consciousness, nor convulsions. All the movements of the tongue are freely executed; no indication of paralysis of the soft palate. These facts are all the more necessary to note, as the speech is thick, nasal at times, as if the tongue was swollen.

Impressions of cold, of pain, of simple contact, of tickling, as perceived as usual in the face, the trunk, the arms, the legs, the soles of the feet. Muscular sense everywhere unimpaired. Neither numbness, nor tingling, nor pains of any sort. No trembling. Muscular strength very considerable everywhere.

For some months L. has been awkward in his hands, and has allowed objects to fall. When he has his eyes closed he is unable to carry his spoon straight to his mouth, or the point of his index finger to the tip of his nose. In the lower limbs analogous

case (2) and Teissier's (4) present features foreign to Friedreich's disease, and ends by saying that an attentive reading of most of the cases which it has been sought to connect with Friedreich's disease shows that authors like Bourneville may have wished to see in this disease a defaced form of disseminated sclerosis. Judson Bury (69, p. 20), thinks too that the cases of Seeligmüller belong rather to disseminated sclerosis, by reason of the trembling, of the exaggeration of the patellar reflexes, and of the nystagmus which they present.

symptoms. In bed, he can execute all movements. . . With eyes open and without any assistance, even a stick, he can go about all day and can ascend or descend stairs. His legs feel stiff, he says, but not weak. Exercise does not tire him. But from time to time he loses his equilibrium, and would fall if he did not find a support within reach.

When his eyes are closed the difficulty of maintaining equilibrium is more conspicuous. He separates his feet to enlarge his basis of support, and throws his arms about in order to balance himself. His movements are not disorderly. His foot alone, moderately raised, falls heavily on the ground a few inches beyond or within the proper spot. He walks more quickly than he wishes to preserve his centre of gravity. At long intervals, when he is strongly exerting himself, cramps and spasms in his legs and toes. Genital functions, micturition, defæcation, normal.

After having left the hospital some months he entered the Hotel Dieu with increased difficulty of walking, which resembles that of individuals affected with locomotor ataxy. *He would be supposed to be drunk.* He separates his legs widely. Persistence of sensibility in its different manifestations—muscular, cutaneous, and of the mucous surfaces. Left on account of *insubordination.*

Topinard adds (4): "MM. Duchenne and Vigla have decided as to the diagnosis in favour of progressive ataxy; we have not the courage to do so. The peculiarities of gait in this case differ too much from what is seen in this disease, and are too identical with those of Case 25 (4), (p. 20) (cerebellar tumour), the diagnosis of which is not doubtful, for us not to maintain reserve."

This reserve was justified. The case without doubt belongs to Friedreich's disease. The youth of the patient, the special characters of the tabeto-cerebellar gait, the spinal curvature, the absence of sensory disturbances, the integrity of the sphincters, the articulatory difficulty, finally the incoordination—everything concurs to corroborate this diagnosis.

Analysis of Certain Symptoms.

The symptoms of Friedreich's disease offer certain peculiarities which deserve to be examined more closely. I shall not make an exhaustive study of them; I shall limit myself to remark upon some among them which appear to me the most important from the clinical point of view.

The ataxy has a quite special feature in this disease, and it is the observations of the Professor of Heidelberg that the partisans of the motor theory of ataxy always quote to demonstrate that sensory disturbances are not at all necessary for the genesis of inco-ordination of movements.

One might be perhaps spared the long discussions which have taken place, and which do not appear to be near termination between advocates and opponents of ataxy by sensory disturbances, if one had at the commencement clearly distinguished the cases of Friedreich's disease from those which belong to tabes properly so-called.

In the latter disease, the theory sketched out in 1862 by Charcot and Vulpian* and developed in the following year by Leyden,† appears to explain pretty clearly the ataxy of tabetics in whom the control of sight supplements at least in part the absence of knowledge of position of limbs, which sensory impressions from the periphery habitually give in the healthy condition. The ataxy is then the result of a lesion in the centrifugal conducting paths, the integrity of which is necessary for the co-ordination of movements. It is a *sensory ataxy*. I shall not pass in review the very serious objections which have been made against this theory, but I will say that it is absolutely insufficient to explain the disorders of movements in Friedreich's disease. There cannot be any question here, in fact, of sensory ataxy. The characters which distinguish the ataxy in this affection are precisely, on the contrary, the absence of disturbances of sensation and of the muscular sense, coinciding with a pronounced and generalised motor inco-ordination.

The following fact, which I have often verified in the patient whose case I was just now reporting, appears to me quite characteristic and capable of furnishing on this question a decisive argument against the sensory origin of ataxy in Friedreich's disease.

I have already related in this case that the patient,

* CHARCOT et VULPIAN, Sur un cas d'atrophie des cordons postérieurs de la moelle épinière, &c., *Gazette hebdomadaire de méd.*, No. xviii., May 2, 1862, p. 281.

† LEYDEN, Die graue Degeneration de hinteren Rückenmarksstränge, Berlin, 1862.

whether his eyes were closed or not, could not touch at the first attempt the tip of his nose or the lobule of his ear with the index finger of one hand when told to do so. Now what appears to me of fundamental importance in order to judge of the nature of the ataxy in this case is that when once his finger touches his face at a certain distance from the point sought, the patient slowly glided his index to the point indicated, *without leaving the surface of the skin*. In thus drawing his finger over the epidermis he evidently guided himself by sensation to remedy his ataxy. The motor incoordination, far from being provoked by sensory disturbances, was on the contrary corrected, thanks to the integrity of the sensory tract.

This character, as well as that of the generalisation of the ataxy, of its propagation from the lower limbs to the muscles of the larynx, of the tongue and of the eyes, prove that the seat of ataxy in Friedreich's disease must be central, placed in the very focus of the centrifugal motor paths of co-ordination of muscular movements—that is to say, in the medullary extension of the cord. It is a *bulbar ataxy*.

Hammond has gone farther. He supposes that the seat of the lesion is in the cerebellum. Most physiologists since Flourens consider the cerebellum as presiding over the functions of co-ordination of movements. Hammond relies on this physiological doctrine to advance the theory that Friedreich's disease has its origin in the cerebellum and the medulla, to be propagated thence gradually to the spinal cord, which is consequently, according to this supposition, secondarily diseased. We shall see, in treating of the pathological anatomy, that the autopsies made hitherto do not allow this view to be adopted. Let us remark, however, from now, that it is contrary to clinical observation, since the symptoms of ataxy are propagated from below upwards, and not from above downwards, as they would be if Hammond's hypothesis had some probability. We have seen, moreover, that the cases of the American Professor do not belong to Friedreich's ataxy, so that the unusual symptoms observed in his patients—vertigo, pains in the back of the

head, &c.—should not be reckoned in the symptomatology of this disease. We shall speak of the choreiform unsteadiness, of the movements of athetosis of the toes, of the articulatory troubles and of the spinal curvature when we shall treat of the diagnosis.

As for *nystagmus*, Friedreich had already made a very profound study of it. He had remarked that the patients had no suspicion at all of their nystagmus, although the oscillations of the ocular globe were very pronounced. Moreover, contrary to what is often observed, the nystagmus in Freidreich's cases was produced when an object was fixed, whilst in the ordinary cases of nystagmus of ophthalmologists, it was then made to disappear, as Professor Seeligmüller has expressly observed in one of his patients. In the case we have described, as we have seen, the nystagmus behaved exactly as Friedreich has described it. We know that the latter writer distinguishes two kinds of ataxy, according as the inco-ordination is observed during movements, or at rest during the continuous contraction of the muscles, which maintains the equilibrium of the body and limbs—locomotor ataxy and static ataxy. The ocular movements also present these two kinds of ataxy.

Nystagmus is a late symptom in Friedreich's disease—one which appears generally several years after the weakness and ataxy of the legs, sometimes even eighteen or twenty years after the onset of the disease. Its importance is great, especially as a diagnostic sign.

The other ocular troubles, so frequent in the tabes of Duchenne, are almost unknown in Friedreich's disease. However, Joffroy ([80]) mentions diplopia and drooping of the upper eylid, and Mendel ([81]) converging strabismus (double paralysis of the sixth pair). In these two cases the pupillary reactions were normal to light, as well as with convergence and accommodation.

Deformity of the foot in Friedreich's disease is a symptom which may exist very early. Rütimeyer relates that they had observed in the families Blattner and Kern the early prominence of the extensor proprius pollicis, which was considered as a sign of very bad augury; the father also

of the children Kern wrote to the doctor that "each time that this traction of the great toe by a sinew" was observed, the parents lost all hope of seeing their child escape the disease.

Friedreich had already observed deformity of the feet in one of his patients, Charlotte Lotsch (his second case): "The group of dorsal flexors of the foot is weakened," says he (the feet are permanently extended by the action of the calf muscles). "*The toes also are permanently flexed,*" and he adds in his second memoir, when speaking of the same case, fourteen years later, "her feet are in a position of permanent equino-varus."

If, therefore, Friedreich has not given a complete description of all the kinds of club-foot which are observed in this disease, we cannot, however, say with Soca that he did not recognise this sign, and we ought to add, in opposition to the statement of Rütimeyer, that he has also proved the existence of dorsal flexion of the toes.

When deformity of the foot is complete, it is accompanied by certain characters which it is important to recognise to differentiate it from other deformities of the feet, especially the tabetic club-foot, as we shall do in the chapter on diagnosis, where we shall more particularly study this deformity.

What distinguishes the club-foot in Friedreich's disease is, first of all, the condition of equinus which results from the predominance of the muscles of the calf over the antero-lateral group of leg muscles, weakened, and even somewhat atrophied. There is indeed a certain degree of foot-drop in these patients, as we have verified in the case we have related. Some authors think that the condition of equinus must be attributed to the contraction of the sural triceps, but we do not experience any resistance in the tibio-tarsal articulation when we raise the foot, and the tendo achillis is not stretched unduly, so that we can readily admit retraction of the calf muscles, but surely not their contraction in the ordinary sense of the word. We must recollect, however, that the contracted muscles behave in a quite peculiar way in tabetics whose reflexes are abolished.

Westphal demonstrated in 1879 that spasmodic phe-

nomena disappear in the combined scleroses, when to the lesion of the lateral columns is added that of the posterior columns, as is the case in Friedreich's disease.

The articulations remain flaccid. Debove has recently quoted an example of it in an ataxic affected with cerebral hæmorrhagé with secondary degeneration of the pyramidal tract, and manifest contracture of the paralysed limbs. Now in this case the reflexes were abolished and the joints remained lax on the hemiplegic contractured side. Might there be analogous contractures in Friedreich's ataxics? That is possible, and then we should have the explanation, not only of the equinus, but also of the dorsal flexion of the toes and of the prominence of the tendon of the extensor proprius pollicis, which also is shortened. This is the opinion of Rütimeyer, who explains also by contracture the deviation of the spinal column.

Be it as it may, one could not interpret in a satisfactory manner the club-foot in Friedreich's disease simply by the contractures of certain muscles. The paralytic weakening of the antagonists of the shortened muscles also enters into it largely in part, if indeed it is not the essential cause of it. It may well be that the claw-like character of the toes may be the consequence of paralysis of the interossei, and the excavation of the soles of the feet that of the relaxation of the plantar muscles. There are still, as may be seen, many obscure points in the genesis of this club-foot. The phenomena which are observed in these feet give to this disease a particular stamp, which separates it clearly from ordinary tabes, in which muscular atrophies and arthropathies, which are complications relatively pretty rare, give rise to deformities of the foot of quite another form, and quite another nature.

In Friedreich's disease the club-foot is a usual phenomenon which is, perhaps, in intimate and direct relation with the central lesion of the columns of the spinal cord. In every case the particular form of the foot is absolutely different from that which M. Dejerine* has recently so well

* J. DEJERINE, Sur l'atrophie musculaire des ataxiques (névrite motrice périphérique des ataxiques), Paris, 1869, and *Revue de médecine*, Feb., March and April, 1889.

studied in his important work on the peripheral motor neuroses of ataxics, in whom muscular atrophy is presented under the form of equinus with plantar flexion of the toes, and particularly of the great toe—that is to say, in a position precisely the opposite of that which is observed in Friedreich's disease.

The *psychical condition* of Friedreich's ataxics deserves special mention. Some authors think that they always suffer from an arrest of intelligence. The case which we relate proves the contrary. I think it is rather in the temperament that anomalies must be looked for. To several patients great irritability of temper is ascribed. Some are apathetic and indolent, others have a caustic turn of mind. We have already spoken of the impulsive laughter which gives such a singular characteristic to the physiognomy.

Diagnosis.

The clinical study which we have just made of the etiology and the symptomatology of Friedreich's disease allows us to simplify very much the discussion of its diagnosis.

In the present state of our knowledge we cannot admit indeed, as belonging to this disease, either the cases with exaggeration of the tendon reflexes (Seeligmüller, &c.), or the forms with paralysis and contractures (Soca). M. Soca says that Friedreich's disease can appear under three different forms, according to the nature of the motor disturbances, which may be of three kinds: 1st, ataxy; 2nd, paralysis; 3rd, contracture. Clinical observation does not, however, authorise us to distinguish these hypothetical classes. M. Soca himself admits doubts on the form with contractures, and in order to prove the existence of the paralytic form he is reduced to quote some cases of Hammond and the case of Kahler and Pick. We have already given the reasons on account of which we could not consider these cases as belonging to Friedreich's disease. We know that this disease presents itself clinically as a well-defined type of ataxy dating from childhood, and before placing under its

title other forms foreign to this type, we must evidently wait till the study of the combined scleroses of the spinal cord is more advanced, which will, perhaps, permit us to connect with Friedreich's disease certain unusual cases which it would be at least premature to actually class in it.

In keeping, therefore, to the clinical type which we have learned to recognise in the preceding chapter, we shall say that the diagnosis of Friedreich's disease is based upon the following characters:

Ataxy and weakness of the legs developing gradually in childhood, almost always without pains, but often simulating choreiform movements. Very slow progressive course, from below upwards, of the disease, which attacks successively the trunk, the arms, the muscles of the larynx, of the tongue, and of the eyes. Ataxic and trembling gait, becoming gradually aggravated. No remissions.

Speech slow, drawling, scanned, as in disseminated sclerosis. Static and dynamic nystagmus. Abolition of knee reflexes. Scoliosis. Peculiar deformity of the foot.

To which must be added negative symptoms which are of very great importance:

Absence of lightning pains. Integrity of cutaneous sensation in all its manifestations. Normal reaction of pupils. Integrity of sight. Absence of genito-urinary troubles.

No syphilitic antecedents. *Family* character of the disease. These are the two principal etiological conditions.

The differential diagnosis will bear essentially on the following affections:—

1. Chorea.
2. Tabes.
3. Disseminated sclerosis.
4. The other combined scleroses of the spinal cord.

I.

Friedreich's disease can be confounded with *ordinary chorea* only at its onset, before the ataxic movements have become sufficiently developed in the lower limbs to interfere with walking. This error of diagnosis, we have already

said, has been made in several cases. We must not, therefore, lose sight of the possibility of a similar mistake, and we shall take care in the cases of chorea which appear suspicious by their manner of walking and their duration, to examine the nature of the ataxic choreiform movements, which are never unilateral in Friedreich's disease, and generally do not invade the facial muscles. We shall assure ourselves also of the presence or the absence of the tendon reflexes, and we shall not neglect to examine the feet, for one of the first signs of Friedreich's disease may be, as we have seen, the dorsal flexion of the great toe, with prominence under the skin of the tendon of its proper extensor.

II.

Locomotor ataxy and Friedreich's disease are distinguished one from another by the most decided characters. Tabes is an affection of adult age; Friedreich's ataxy a disease of childhood. It would not, however, be necessary to go so far as to say with M. Soca: "A child or a youth, who is ataxic is a case of Friedreich's disease. This is not absolutely true, but it is sufficiently exact for practical purposes."

Although tabes of adolescents may be a rare disease, there exists, however, now a sufficient number of authentic cases to invalidate this proposition. On the contrary, it is very necessary to guard against making a like diagnosis; and before admitting that an ataxic child may be affected with Friedreich's disease, it is necessary to search with much care for the presence of the symptoms which characterise this disease. We know that all kinds of cases of infantile ataxy have been too readily attributed to Friedreich's disease, without selection and without criticism, at the risk of spoiling the clinical picture of it.

The tabes of children, if we are to judge of it by the cases which have been published, does not differ from tabes in the adult. The ocular symptoms, the sensory disturbances, the lightning pains, the paræsthesiæ, the gastric crises, and the

urinary troubles, furnish sufficient differential characters to prevent any hesitation in the diagnosis. The importance of syphilis as an etiological antecedent has quite recently been emphasised by a case of Professor Strümpell at Erlangen. It relates to a little girl with hereditary syphilis, who was affected with tabes and general paralysis at the age of thirteen years.* The same holds good in the case of the little patients of E. Remak,† who suffered from hereditary syphilis, and all three presented atrophy of the optic nerve without ataxy. Dr. Berbez,‡ who relates fourteen cases, concludes that in early tabetics, heredity is strong, and that intensity of the disease and multiplicity of symptoms are the necessary accompaniments of early tabes. He remarks also after the teaching of M. Charcot, that, although essentially hereditary, early ataxy has nothing in common with the hereditary ataxy of Friedreich.

MM. Gombault and Mallet§ have quite recently published a case of tabes occurring in childhood, with an autopsy, the report of which appears to us particularly instructive for the discussion of the differential diagnosis of Friedreich's disease and locomotor ataxy. Here is a *résumé* of this report.

Man, aged fifty-seven, infirm from childhood, died a few months after entering the hospital. The patient has, therefore, been only a very short time under observation at the close of his life. Inquiries as to etiology are almost entirely wanting. His father died when the child was seven years old and was already infirm. There is no information as to him. His mother died of acute mania. The patient was placed in a hospital because, it was said, he was permanently insane, and refused to take food. All that we can learn of his history is that the onset of his disease dated from childhood, that he was not seven years old when he fell ill, and that at ten years of age he was definitely admitted as

* A. Strümpell. Progressive Paralyse mit Tabes bei einem 13 jährigen Mädchen. *Neurologisches Centralblatt*, March 1, 1888, no. v., p. 122.

† E. Remak, Drei Falle von Tabes im Kindesalter. *Berliner klinische Wochenschrift*, 1885, no. vii., p. 105.

‡ P. Berbez, Tabes précoce et hérédité nerveuse. *Progrès médical*, no. xxx., July 23, 1887, p. 59, *cf.* W. Jacubowitsch, Tabes dorsalis im Kindesalter, *Archiv für Kinderheilkunde*, V Band, 1884, p. 187.

§ Albert Gombault et Mallet. Un cas de tabes ayant débuté dans l'enfance. Autopsie. *Archives de médecine experimentale*, first year. no. iii., May, 1889, p. 385.

a patient in an asylum. From that period continuous and progressive course of the disease, which consists principally in disturbance of the voluntary movements, involving the four limbs. The patient could not maintain the erect position unless he held on to the furniture, and kept looking at his feet. He fell if his eyes were closed. At the time of admission to the infirmary flaccid paralysis of the lower limbs; absence of tendon reflexes; motor inco-ordination of the upper extremities; inability to appreciate the position of the limbs; muscular atrophy; muscles of the thigh and of the leg altogether atrophied; ape-like hands; pronounced scoliosis, the date of development of which cannot be stated, because the patient does not remember it. Did it exist in childhood? Diplopia; slight nystagmus; profound disturbance of sensibility in all the limbs; manifest retardation of tactile and painful sensations; anæsthesia; analgesia; thermo-anæsthesia; sensibility is intact only in the face; no trembling; no brain disturbance; no incontinence of urine or of fæces; pains from time to time, not definitely of the lightning character.

Talipes equino-varus of the bedridden; white and soft œdema of the malleoli; speech distinct and easy; no hallucination; penis and testicles fully developed; scar on the sacrum; death.

Autopsy.—Calcareous plates in the arachnoid, all along the cord at its posterior part, especially in the dorso-lumbar region. Issuing from the lumbar enlargement, the posterior roots are bulky, translucent, gelatinous, nodulated, covered with an abundant meshwork of vessels. The anterior roots also are very much hypertrophied. Spinal cord altogether bulky; grey condition and gelatinous appearance of the posterior columns. Peripheral nerves bulky without nodosities, vascular, rose-coloured, translucid.

The grey matter is much reduced in size, atrophied, and its shape has altered. It is thin and delicate in the lumbar region, which one would take at first sight for a section of the cervical region. Antero-lateral columns relatively large. They are furrowed by connected perivascular tracts larger than in the normal condition. Posterior columns small, and stained of a rose colour by carmine, especially at the inner part of the posterior zones. Sclerosis of the posterior columns. In the posterior cornua diminution, almost disappearance, of the bundles of fibres belonging to the posterior roots. In the anterior cornua diminution in number and atrophy of the large motor cells.

Vesicular column of Clarke normal in the dorsal and dorso-lumbar region. The pia mater is everywhere notably thickened.

In the cervical region, sclerosis of the columns of Goll; the posterior root zones are sclerosed in this region only here and there.

Hypertrophic sclerosis of the peripheral nerves is much more developed than in the spinal roots. An almost complete disappearance of the myeline has been observed in the peripheral nerve-tubes which have been examined.

In the discussion of this case, the authors say that it is hardly necessary to point out the characters which differentiate it from Friedreich's disease. However, when we read the description of symptoms which they give, we cannot help thinking that the patient of MM. Gombault and Mallet presented in the course of his disease several symptoms absolutely analogous to those of Friedreich's disease:—motor inco-ordination commencing in childhood; abolition of tendon reflexes; course of the affection slow, unobtrusive, continuous and progressive, attacking the four limbs, without pains at the commencement; talipes equino-varus. Have we really in this case the club-foot of the bed-ridden, as some authors think? Deformity of the vertebral column:—MM. Gombault and Mallet remark in reference to this deformity, that it frequently accompanies various generalised affections which have commenced in childhood. But we have now to find out the differential diagnostic signs which allow us to distinguish this particular case from Friedreich's disease, and scoliosis is precisely a character common to the two cases, the importance of which must not be minimised. Nystagmus:—it is true that the authors quote the absence of nystagmus (*loc. cit.*, p. 403) as contributing to exclude Friedreich's disease in their case. They have forgotten that some pages before (*loc. cit.*, p. 387) they themselves have expressly mentioned the existence in their patient of a "slight nystagmus." Finally, the integrity of the sphincters: That is quite as many symptoms as one could refer to Friedreich's disease. And when the authors affirm that the complete absence of cerebral symptoms (mental symptoms being necessarily put on one side) bears witness in their case against Friedreich's disease, they forget again that they have mentioned diplopia (*loc.*

cit., p. 387) in the antecedents of their patient. What, then, are these cerebral symptoms which characterise "the now classical description of Friedreich's disease"? It is precisely, on the contrary, in the absence of these symptoms that we must find a character of the first order to distinguish this disease from ordinary tabes. The authors add, it is true, "and in particular, absence of articulatory troubles," showing thus that they include these troubles among the cerebral symptoms. But those with nystagmus are the only really classical bulbar symptoms which belong incontestably to Friedreich's disease!

We must seek elsewhere for the fundamental reasons which do not allow us to admit Friedreich's disease in the case of MM. Gombault and Mallet. They are especially the generalised muscular atrophies, and the intensity and the extent of the sensory disturbances. These are the essential symptoms which absolutely exclude, in this case, Friedreich's disease.

The authors, who found at the autopsy the peripheral nerves more diseased than the spinal roots, put forward the fairly probable hypothesis of the possibility of a polyneuritis followed by an affection of the cord. Perhaps, say they, we have to deal in this case with multiple peripheral primary neuritis, with consecutive involvement of the spinal cord in the course of development. They arrive, however, at the provisional conclusion, "that we must consider the case as an example of locomotor ataxy of spinal origin, developed in early age."

There is a hypothesis which pleases us more, and we have been struck by not seeing it expressed in the interesting work of MM. Gombault and Mallet. We shall willingly admit in this case a syphilitic affection, perhaps congenital. We know how frequent syphilis is in the hereditary antecedents of tabetic children—we have just given several examples of it. It would therefore be quite possible that syphilitic infection may have also played its part in the patient of MM. Gombault and Mallet. We must not forget either that there existed in this case the remains of a very pronounced anterior poliomyelitis, which also complicated the symptomatology of it.

Let us return to the differential diagnosis of locomotor ataxy and Friedreich's disease, and let us prove first that the symptoms which they offer in common, when examined more closely, are not at all alike.

The ataxy itself presents considerable differences in the two diseases, and the footprints show that the gait is far from being identical in tabes and in Friedreich's disease. In the latter affection the patient progresses in a very irregular zig-zag manner, instead of keeping in a straight line as tabetics do. Nothing displays better than the method of taking footprints, the fundamental difference which exists between the clubfoot of Friedreich's disease, and the deformities of the foot in tabes. The tracing of tabetics affected with arthropathies of the feet shows the print of flat feet, whilst that of the clubfoot of Friedreich is characterised by the complete absence of the print of the sole of the foot corresponding to the excavation which we have described. M. Joffroy (86 and 103) judiciously remarks, moreover, that the deformity of the foot is never developed in tabetics, except when the patient has been for some time confined to bed, whilst in Friedreich's disease the deformity of the foot exists in the patient who is able to walk, long before he is confined to bed.

Romberg's sign discloses also, as we have observed, a fundamental difference in the two diseases. Whilst in tabes closure of a patient's eyes makes him lose his equilibrium, because he has no longer knowledge of the position of his limbs, in Friedreich's disease it is in consequence of choreiform instability. The patient sways whether his eyes are opened or closed, but in the latter case having lost the control of vision, the swaying movements easily pass the limits of equilibrium, and he falls, although he may have preserved the notion of the position of his limbs.

Must we consider, with M. Joffroy, diplopia as a symptom that may belong to the picture of Friedreich's disease (103). We should not dare to speak affirmatively on this point. Diplopia, indeed, in M. Joffroy's cases has been transitory. It is noted in the history of the patient, who belonged to a tuberculous family, but it was not directly observed, we

think, in the medical examination. In every case it does not appear to have presented the lasting characters which it sometimes manifests in tabes. Transitory diplopia is met with in a great number of nervous affections. It is not rare in certain neurasthenics. It has been several times seen in cases of poisoning by tainted meat. Prof. Bouchard* and many other authors have pointed it out in dilatation of the stomach, in which we have many times proved it. It would not, therefore, be right to conclude from its presence in the history of a patient suffering from Friedreich's ataxy that it necessarily forms a part of the assemblage of symptoms in this affection. We must wait for further observations before admitting diplopia as a definite symptom of Friedreich's disease, of the same importance as in locomotor ataxy. And even, if it ever is admitted as such, it will not remain less exceptional in the first, whilst it is usual in tabes.

We shall not insist any further on the differential diagnosis of these two diseases, which is now well known, and does not offer any difficulty in ordinary cases.

A word only on nystagmus, very frequent as is known in Friedreich's patients, and which was thought never to exist in tabetics. It is observed, however, very clearly in some cases of tabes, but it is true that these cases are very rare. M. Gilles de la Tourette has published three cases of it. I have seen it myself in two cases, one of which is briefly reported in the note on my observations on suspension in tabes.†

In the following comparative table is a résumé of the differences which exist between the two diseases, the symptoms being put parallel :—

Friedreich's Disease.	Tabes.
"Family" character. Several children are usually attacked in the same family.	Rarely more than one tabetic in the same family.
Scarcely ever syphilis in the antecedents.	Syphilis is usually found in the history, either acquired or hereditary.

* Ch. Bouchard, Leçons sur les auto-intoxications dans les maladies. Paris, 1887, p. 174.

† Ladame, De la suspension dans le tabes. *Rev. méd. de la Suisse rom.*, June 20, 1889, p. 347.

The disease begins in childhood. There are no authentic cases known of onset after the 20th year.	Onset almost always after the 20th year. Infantile tabes is most often of syphilitic origin.
No lightning pains either at the onset or during the course of the disease. They have been only exceptionally observed, and in these cases are they really lightning pains?	Lightning pains perhaps preceding ataxy for several years. Scarcely ever absent in tabes.
No preataxic stage. The commencement of the disease may simulate a slight chronic chorea for several years.	Long preataxic stage, presenting the most diverse symptoms: ocular, visceral, &c.
Ataxy during rest and during movement. Choreiform instability. Movements incoordinate, feeble and not sudden. Ataxy is general. It progresses slowly from below upwards. Gait, tabeto-cerebellar, ataxic and festinating. Closure of eyes causes loss of equilibrium only by reason of the choreiform instability, due to static ataxy.	Ataxy during movement. At rest the patient is quiet. Ataxic movements violent, sudden, as of a spring which is let go. The ataxy may be localised in the legs or in the arms (cervical tabes). Gait plainly tabetic. The patient follows a straight line. Closure of the eyes causes loss of equilibrium, because it takes away the knowledge of the position of the limbs (Romberg's sign).
Integrity of sensation in all its manifestations. Towards the end of the disease only, in some cases, slight affections of the cutaneous sensation.	Anæsthesia and hyperæsthesiain patches. Paræsthesia. Retardation of transmission of painful sensations and other perversions of sensation.
Absence of knee reflexes. Cutaneous reflexes normal. Plantar reflexes rather exaggerated on account of the great sensibility of the soles of the feet.	Absence of knee reflexes. Cutaneous reflexes often weakened or abolished on account of affections of sensation; the plantar reflexes especially are frequently absent.
Integrity of the special sense organs. Horizontal nystagmus, static and dynamic, is rarely absent. Very rarely affections of the muscular innervation of the eyes.	Grey induration of the optic nerve. Amaurosis. Sclerosis of the auditory nerve. Buzzings in the ears. Menières' vertigo. Myosis and Argyll-Robertson sign. Pareses of muscles of the eye; diplopia, ptosis. Scarcely ever nystagmus.
Absence of visceral affections.	Laryngeal crises (spasms of the glottis). Gastric, intestinal, anal, vesical, nephritic, crises.

No lesions of the skin, the joints or the bones. Club-foot of special kind, habitual, in consequence of pareses and muscular contractures. Athetosis movement of the toes in walking. No peripheral neuritis.	Cutaneous lesions (zona). Trophic affections of the nails. Shedding of the teeth. Spontaneous fractures. Arthropathies. Tabetic foot. Muscular atrophies. Peripheral neuritis, sensory and motor. Other trophic troubles. Perforating ulcer of the foot.
Integrity of the genito-urinary functions and of the sphinters.	Genito-urinary troubles. Impotence. Paresis and hyperæsthesia of the bladder. Retention and incontinence of urine. Constipation.
Scoliosis.	No spinal curvature.
Speech slow, scanned.	No speech troubles.
Progressive, uniform, very slow development of the disease, without crises or prominent episodes.	Pretty often relatively rapid development of the disease by successive crises, followed by obvious aggravation, with numerous fresh symptoms from time to time.

III.

In the memoir of Marie,‡ and in the more recent one of Unger,* will be found very complete bibliographic notices on *disseminated sclerosis* in children, to which we shall add the work of Professor D'Espine, of Geneva.† It is seen from these works that disseminated sclerosis most often presents in children the cerebro-spinal type which commences by convulsions. The only positive symptoms which may be common to this affection and to Friedreich's disease are the nystagmus and the slow and scanned speech. But these troubles are late in Friedreich's disease, and only appear long after the ataxy, the loss of knee reflexes and the other symptoms which cannot leave any doubt as to the diagnosis,

‡ PIERRE MARIE, De la sclérose en plaques chez les enfants. *Revue de Médecine*, 1883, p. 536. *See also* MONCORVO, Contribution à l'étude de la sclérose multiloculaire chez les enfants, Paris, 1884.

* L. UNGER in Wien, Ueber multiple inselförmige Sclerose des Centralnervensystems im Kindesalter, Leipzig und Wien, 1887.

† A. D'ESPINE, Deux formes de paralysies chez les enfants. *Revue médicale de la Suisse romande*, March 20, 1889, No. 3, p. 129. *See also* D'ESPINE et PICOT, Manuel des mal. de l'enf., fourth edition, p. 441.

whilst the exaggeration of the reflexes, the rigidity of the legs, the contractures, the trembling on voluntary movement, the disturbances of the motor innervation of the eyes, diplopia, nystagmus, strabismus, &c., are the first signs of disseminated sclerosis. Moreover, all the other symptoms so characteristic of disseminated sclerosis, apoplectiform and epileptiform attacks, vesical paralysis, trophic and mental disturbances, frequent remissions in the course of the disease, all these are invariably wanting in Friedreich's disease, as this author himself had already plainly stated.

Friedreich's Disease.	Disseminated Sclerosis.
Family character of the disease. Insidious onset.	Case isolated in a family. Sudden onset, often with convulsions.
Very slowly progressive course of the affection. No remission of symptoms.	Frequent remission of symptoms. Irregular course of the disease.
Choreiform instability. Athetosis movements of the toes. Paresis of legs, with loss of tendon reflexes. Progressive ataxy of the four limbs.	Trembling on voluntary movement. Spastic paresis. Exaggeration of tendon reflexes. Ankle-clonus. Contractures.
Tabeto-cerebellar gait. Integrity of special sense organs and of the ocular muscles. Late nystagmus.	Spastic gait. Paralysis of ocular muscles. Diplopia. Amblyopia. Early nystagmus.
Scoliosis. Special club-foot. integrity of genito-urinary organs and of the sphincters. No visceral tabetic troubles.	Congestive, apoplectiform and epileptiform attacks. Vesical paresis and other tabetic symptoms, generally slightly marked. Mental disturbances.

Characters Common to the two Diseases.

Absence of sensory disturbances, slowness of speech, scanning of words. Nystagmus.

Vertigo has also been noted as a symptom common to the two diseases, but we do not yet know if vertigo really belongs to this affection, although it may often have been mentioned in reports, since the first case described by Friedreich. Our patient has never experienced vertigo.

IV.

Combined Sclerosis of the Spinal Cord.‡ We shall not undertake here the study of the subject of the combined sclerosis, which is, perhaps, really the most obscure of spinal pathology. From the anatomical point of view, Friedreich's disease is a combined system sclerosis of the posterior and lateral columns, which could not, in many cases, be distinguished, as regards its spinal localisation, from other combined sclerosis, having a like anatomical seat, with very different clinical symptoms. It is evident that the special evolution of the lesions, their variable extent and the successive separate invasion of different systems of fibres, have a decisive importance for the symptomatology. Friedreich's disease which evolves on an incompletely developed cord, forms a very distinct group, and henceforth is classed among the combined system scleroses.*

In spite of the anatomical lesions which it presents, we yet do not think, with Professor Grasset, that Friedreich's disease can be put in the category of "combined tabes." We think it would be preferable to reserve this appellation for the cases of tabes which are complicated by a diffuse sclerosis of the lateral columns, most often following a posterior spinal meningitis, meningo-myelitis by propagation, as Friedreich and M. Dejerine† have proved it. As for the combined scleroses properly so-called of the postero-lateral columns, with primary system lesions of the posterior columns and pyramidal tracts, we actually possess a number of cases of them sufficient to sketch out the

‡ *See also* J. L. PREVOST, Sclérose des cordons postérieurs, compliqué d'une sclérose systématique des cordons latéraux. *Archives de Physiologie*, 1877, t. iv., p. 764. V. BABESIU, *Virchow's Archiv.*, 1879, vol. lxxvi., p. 74. EDES, *Boston Med. et Surg. Journal*, September 21, 1882. RAYMOND, *Arch. de Physiol.*, 1882, x., p. 456. DAMASCHINO, *Gazette des Hôpitaux*, No. 1, 1883. HOPKINS, BRAIN, October, 1883. ORMEROD, id. April, 1885. BABINSKI et CHARRIN, *Revue de Méd.*, 1885, November, p. 962. MASSALONGO, *Medic. Contemp.*, October and November, 1886. BORGHERINI, *Riv. sper. d. Freniat*, XIII., p. 137, 1887, &c., &c.

* Among recent treatises that of Professor L. HIRT of Breslau (*Pathologie und Therapie der Nervenkrankheiten*, Wien, 1890, p. 364), is to our knowledge the only one in which Friedreich's disease is separated from tabes and placed among primary combined scleroses.

† DEJERINE, *Archives de Physiologie*, 1884, iv., p. 456; *Semaine Médicale* 1886, No. 18, p. 181.

symptomatology of them. There exist, doubtless, very considerable differences in the symptoms described by observers in the different cases that we know. Nevertheless, we can provisionally distinguish from the clinical point of view, four or five categories of patients affected with combined scleroses, which belong to the following types (without mentioning syphilitic myelitis and others, which give rise to secondary scleroses more or less diffused, and which may simulate system scleroses) :—

I. *Ataxic paraplegia* (Gowers‡). In this we may have all the usual symptoms of tabes with predominance of paralytic weakness of the legs belonging to sclerosis of the lateral columns. If the lesion of the posterior columns stops in the dorsal region, or even if it descends in the lumbar region without attacking the posterior root zones, the knee reflex will be intact, or even exaggerated, in consequence of the sclerosis of the pyramidal tract (Westphal*).

II. *Spastic tabes dorsalis* (Charcot, Erb, Strümpell). On several occasions combined sclerosis of the postero-lateral columns has been discovered at the autopsy of patients having presented the clinical picture of spastic spinal paralysis, whenever sclerosis of the pyramidal tracts involved all the lumbar region.

III. *Ataxo-spastic tabes* (Grasset†). A mixture of the symptoms of locomotor ataxy of Duchenne with those of spastic tabes.

IV. *General paralysis.* Westphal has shown that primary sclerosis of the lateral columns combined with system sclerosis of the posterior columns is especially frequent in general paralytics. According to this observer primary

‡ R. GOWERS, Clinical Lecture on Ataxic Paraplegia. *The Lancet*, 1886, vol. ii., p. 1, 61 and 130.—DANA, Progressive spastic ataxia (combined fascicular sclerosis) and the combined scleroses of the spinal cord. *The Medical Record of New York*, 1887, July 2, vol. xxxii., No. 1, p. 1. DANA, Ataxic Paraplegia, BRAIN, January, 1889, p. 490.

* WESTPHAL, *Virchow's Archiv*, Band XXXIX. and XL.; *Archiv. für Psychiatrie*, Band V., p. 803, Bd. VIII., p. 469, Bd. IX., p. 413 and 691, Bd. XV., p. 224. *See also* STRÜMPELL, Ibidem, Band XI., p. 27, Band XVII. (1886), p. 217. DEBOVE, De l'hémiplégie des ataxiques, *Progrès Médical*, 1881. No. 52, p. 1021, No. 53, p. 1042.

† GRASSET, *Archives de Neurologie*, 1886, t. xi., p. 156 and 380, t. xii., p. 27. Cf. BALLET and MINOR, Ibidem, 1884, t. vii., p. 44.

isolated sclerosis of the lateral columns has hitherto been certainly observed only in general paralysis.‡

We have no need to go into the arguments which allow us to make the differential diagnosis between Friedreich's disease and the combined scleroses of the spinal cord which belong to the four preceding groups. This diagnosis can offer no difficulty.

It is not the same with a final group in which may be included all the cases which do not enter into the above categories. But it is here precisely that investigations and authentic cases are wanting. Some facts might raise a suspicion that there exists perhaps a form of Friedreich's disease peculiar to adult age; but we are still reduced on this point to a simple hypothesis which requires confirmation. Under these circumstances there can be but one course to follow. We must keep rigorously to the symptomatology of Friedreich's disease such as we know it, with its hereditary characteristics, and eliminate from it as we have done, all the cases which do not exactly correspond with it. The analogies, still very obscure, of pathological anatomy, ought not to serve us as a guide in these questions, but much rather clinical observation, till a deeper study of the combined scleroses, system or diffused, will better enable us to enlighten ourselves in these delicate and complicated researches of spinal pathology.

The report presented by Dr. Oppenheim at the meeting of the Society of Psychiatry of Berlin, Nov. 12, 1888, is particularly instructive on this point.* It is a case of combined sclerosis of the spinal cord in a child aged 15 years, who suffered from a progressive affection of sight from the age of 10 years, after measles, but whose other spinal symptoms only appeared at the age of 12 years, after a fall in the water. At first choreiform movements in the left half of

‡ ZACHER, Beiträge zur Pathologie und pathologischen Anatomie der progressiven Paralyse. *Archiv. für Psychiatrie*, Bd. XIV. 1883, p. 463, Bd. XV., 1884, p. 359. SIOLI, Ein Fall von combinirter Erkrankung der Rückenmarkstränge mit Erkrankung der grauen Substanz. *Archiv. für Psych.*, vol. xi., 1881, p. 633.

* OPPENHEIM, Ueber einen Fall von combinirter Erkrankung der Rückenmarkstränge im Kindesalter. *Neurologisches Centralblatt*, December 1, 1888, No. 23, p. 647.

the body, especially in the face and the left arm, then weakness and slight contracture in the joints of this arm. A second group of symptoms is allied to the clinical picture of tabes. Double optic atrophy. Pupils fixed. Ptosis on the right side. Absence of knee reflexes. Weakness and slight ataxy of the limbs. Romberg's sign. No sensory nor sphincter troubles. Death from bedsore. Sclerous atrophy of pyramidal tracts and of the columns of Goll and Burdach. The lesion of the posterior columns was less marked than that of the lateral columns.

We see from the above description of symptoms how easy it was to exclude Friedreich's disease in this case. Oppenheim's case shows that there exists a combined sclerosis of the postero-lateral columns in children with symptoms in great part tabetic, and essentially different from those of Friedreich's disease. This case would be sufficient to distinguish clearly the latter disease from the combined scleroses, and to give it a place apart in the primary system lesions of the spinal cord, in spite of the anatomo-pathological analogies which bring it near to other combined scleroses.

Pathological Anatomy.

The number of autopsies which have allowed us to fix the anatomical lesions of Friedreich's disease is still very restricted, and if one can say henceforth that these lesions belong to the group of primary combined system scleroses, there yet remains no less obscurity on their nature and their precise localisation.

We have altogether nine autopsies of authentic cases, of which five belong to Friedreich's patients, one to Everett Smith, one to Newton Pitt, and two to Rütimeyer.

Friedreich (11), who had himself made the autopsy on four of his patients, arrives at the following conclusions :—

The "hereditary" character of the disease is explained, says he, by an arrest of development of the spinal cord. The volume of this organ was very much reduced in all the cases in which an autopsy was made. The spinal cord was

slender, flattened, and as if atrophied in its entirety. It was not difficult to convince oneself that the bulb, and also the cord, had not reached their complete development, so that the sclerosis degeneration of the posterior and lateral columns had invaded an organ still incompletely developed. This is confirmed by the discovery of Dr. Schultze, who found, on microscopical examination, the nervous elements of the healthy portions of the cord very small and very slender.

At the first autopsy Friedreich saw only sclerosis of the posterior columns. The anterior and lateral columns are noted as healthy. In the three others the invasion of the lateral columns by the sclerosis has been clearly determined. But what especially struck Friedreich is the posterior spinal meningitis, which is the true cause, according to this author, of the propagation of the chronic inflammation from the posterior columns to the lateral columns. That is why, says he, in the lower portions of the cord, where the meningitis is more recent, the margin of the lateral columns is less degenerated than in the cervical portion, where all the periphery is degenerated like a ring. We know now that this explanation of Friedreich has no longer much value. In fact, the difference which he had proved between the extent of the cortical degeneration in the lumbar and cervical regions proceeds from this, that in the latter the direct cerebellar tracts are degenerated; there we have a primary system sclerosis which forms part of the combined lesions of Friedreich's disease, the significance of which was completely ignored at the time of the first autopsies made by this eminent observer.

It is true that in Friedreich's cases, and especially in the one, the autopsy of which was made by Schultze (see later), the cortical degeneration went appreciably beyond the region of the direct cerebellar tracts. But the lesions of the cord were examined in these cases only after the disease had lasted twenty-three and thirty years, whilst in Rütimeyer's cases, the duration of which was only thirteen and nine years, the lesions were confined, as we shall see, to these tracts. We must therefore suppose that in Friedreich's

cases the sclerosis has extended itself, and has gone beyond its first limits owing to the long duration of the disease, so that the diffuse cortical degenerations may be regarded as accessory, having slowly developed themselves, perhaps under the influence of the slight spinal meningitis, and at length complicating the primary system changes.

The cells of Clarke's columns are in intimate relation with the direct cerebellar tracts, of which they represent the trophic centres. Now Friedreich had already remarked that there existed in one of his cases a double canal in the dorsal region of the cord. This double canal, says he, is probably nothing else than the degeneration of Clarke's columns, a degeneration verified in all the other cases, in which the number of Clarke's cells was very much diminished, sometimes even reduced to a solitary one in the lower dorsal portion, that is to say in the region in which they are usually most developed.

A fifth patient of Friedreich was examined post-mortem by Dr. F. Schultze ([20]), who found lesions similar to the preceding ones: grey degeneration of the posterior columns and of the posterior portions of the lateral columns; flattening of the cord from before backwards; slight thickening of the pia mater. Small size of the healthy portions of the cord. As a difference no circular complete cortical degeneration in the upper cervical region, but degeneration of the inner parts of the anterior columns along the longitudinal fissure extending up to the pyramids. Degeneration of the columns of Clarke. Posterior roots atrophied; integrity of the ganglia of the medulla oblongata.

Everitt Smith gives drawings of sections of the spinal cord of his patient, and describes the anatomical lesions as follows:—

Spinal meninges injected and adherent to the bony walls of the canal. The cord presents an asymmetric atrophy, it is pale and softened in places. The dorsal and lumbar regions, carefully examined, show marked sclerosis of the posterior columns in their whole extent, with the exception of a small part, relatively healthy immediately behind the posterior commissure. Sclerosis of the direct and crossed

pyramidal tracts less intense than in the posterior columns. Thickening of the pia mater; atrophy of the posterior roots; the anterior normal. Ganglion cells of the anterior and posterior cornua less numerous than normally, and showing in a high degree degenerative change.

Numerous figures demonstrate the seat and extent of the combined sclerosis in Dr. Pitt's case (74), the autopsy of which is described with the most minute details. The author gives a series of comparative measurements of a healthy cord and of the diseased cord, which prove how much smaller the sections of the latter were than those of the other throughout the length of the cord. Posterior roots very slender also, especially in the cervical and lumbar enlargements. Nowhere opacity or thickening of the meninges, which were found completely normal. Grey degeneration of the posterior and lateral columns. The hardened cord was examined by Weigert's method. We shall not reproduce the interesting details which Dr. Pitt gives of each region examined, we shall limit ourselves to presenting a *résumé* of his conclusions (*loc. cit.*, p. 389).

I. The spinal cord is extremely slender.

II. Extreme sclerosis of the columns of Goll in their whole length from the lumbar enlargement to their termination in the floor of the fourth ventricle.

III. Intense sclerosis of the posterior part of the columns of Burdach, in which, however, some healthy fibres are scattered, in greater number at the upper part of the cord.

IV. The zone of the columns of Burdach, which bounds the cornua and the posterior root, is intact. (This region bears now the name of marginal zone of Lissauer).

V. Sclerosis not distinctly limited (much less intense than in the posterior columns) of the crossed pyramidal tracts.

VI. Sclerosis of the ascending cerebellar tracts, visible up to the decussation of the pyramids. Very slight and irregular sclerosis of a few scattered fibres in the antero-lateral columns, especially at the periphery, and in some sections along the anterior fissure.

VII. Degeneration of the columns of Clarke in some sections.

VIII. Degeneration of some fibres of the posterior roots, and of the posterior cornua.

IX. Friability and shrivelling of the affected regions.

What was most striking to the naked eye was the great diminution of the transverse section of the cord, as well marked in the cervical enlargement as in the dorsal region and in the lumbar enlargement, the posterior portions being proportionally more diminished than the anterior.

Columns of Goll extremely sclerosed. Numerous healthy tubules scattered in the columns of Burdach and in the anterior third of the posterior columns. A fasciculus almost intact along the posterior cornua. Sclerosis of the crossed pyramidal tracts, and of the direct cerebellar, less pronounced. A slight annular degeneration extends from the periphery to the anterior part of each side of the anterior fissure.

The most marked sclerosis is always in the posterior columns. Posterior nerve roots in part degenerated. In the lateral columns the sclerosis is limited to the crossed pyramidal tracts, and to the direct cerebellar tracts. No degeneration around the posterior commissure. Clarke's columns are sclerosed.

Posterior columns almost completely sclerosed. Anterior quite normal. The triangular sclerosed patch of the lateral column corresponds exactly with the crossed pyramidal tract in this region. It is separated from the posterior root by a band of healthy tissue (marginal band of Lissauer).

Dr. Pitt remarks finally on the frequency among these patients of cardiac troubles, which have not yet been noticed by authors, who give for the most part little attention to abnormal conditions of the circulation and of the blood-vessels, which are frequently degenerated, according to Pitt, in Friedreich's ataxics.

In favour of an association of Friedreich's disease with a tendency to vascular degeneration and to the imperfect development of the spinal cord, Dr. Pitt draws attention to the following points:—

1. That the posterior columns which are the most vascular, have always been found smaller and more atrophied than the rest of the cord;

2. That vascular lesions have been demonstrated in several cases, and that the disease has always been much aggravated after acute intercurrent affections;

3. The development of the cord begins in the most vascular regions, so that the degenerations attack by preference the parts which are developed in the last place. These parts have also the least important functions, and, according to the general law of Hughlings Jackson, these are the first which degenerate;

4. The sclerosed regions correspond to the tracts, the filaments of which are covered with myeline after the 5th month of fœtal life;

5. The symptoms become more marked at the time of puberty, which is accompanied, as we know, by great vascular modifications in the organism.

The two most recent autopsies are those of Dr. Rütimeyer, of Bale ([77]). By the courtesy of this distinguished writer I have been able to examine with him the interesting series of sections which he has made in his two cases. I can, therefore, speak with authority, and I embrace this opportunity to address again my thanks to Dr. Rütimeyer. The following is a résumé of these two autopsies:—

1. H., age twenty years, died rather suddenly of general weakness after 13 years' illness. Medulla oblongata and spinal cord very much atrophied throughout. Thickening of the meninges and multiple adhesions, especially in the lumbar region. Posterior roots very much thinned. Firm consistence. With the naked eye grey degeneration of the column of Goll is very well seen in the upper cervical region, and of the columns of Burdach, and of the posterior portions of the lateral columns in the cervical enlargement and the dorsal region. This degeneration corresponds exactly in the lateral columns with the direct cerebellar tracts and the crossed pyramidal tracts. It is clearly separated from the posterior cornu on either side by a narrow zone of healthy white matter. It is in the lower dorsal region that the scleroses attains its maximum intensity, as well in the lateral as in the posterior columns. In the upper lumbar

region the middle fibres of the radicular zone are much degenerated, which involves the abolition of the knee reflexes during life, as the researches of Westphal have shewn. Absolute integrity of the marginal zone of Lissauer, and of the grey substance generally; whilst in the dorsal region the columns of Clarke, in the grey matter, are degenerated and their cells atrophied. This atrophy is at its maximum in the lowest dorsal region, where ganglion cells are no longer to be found.

Symmetrical degeneration of the lateral columns, strictly limited to the direct cerebellar and crossed pyramidal tracts. Maximum scleroses in the medium portions of Goll's columns.

Almost complete degeneration of the posterior root zones. The degeneration of the lateral column corresponds exactly with the section of the crossed pyramidal tract in this region.

2. Girl, age 14, ill for nine years, died of general debility following typhoid symptoms. Spinal cord very slender, especially in the dorsal region. The same symmetrical lesions of degeneration in the posterior and lateral columns (crossed pyramidal and direct cerebellar tracts) as in the first case. The medulla of extremely small dimensions. Microscopic examination, made with the greatest care, as in the foregoing case, reveals the same sclerous degenerations in the different regions of the spinal cord. In the grey matter of the dorsal region the columns of Clarke are much degenerated; also the posterior roots. Moreover, the posterior cornua are inact. The same integrity of the zone of Lissauer as in the first case.

Intense degeneration of the columns of Goll. Degeneration of the columns of Burdach reaches to the neighbourhood of the posterior columns from which it is separated by an intact zone. In the lateral columns the sclerosis involves exactly the section of the direct cerebellar and crossed pyramidal tracts.

The maximum sclerosis is always in the columns of Goll, next in those of Burdach. The degeneration of the lateral columns is less pronounced. A slender band of normal

tissue borders the posterior cornua. The antero-external zone of the posterior columns is normal. The degeneration of the lateral columns extends in front beyond the limit of the cerebellar tracts. Is this an individual anomaly or a marginal cortical sclerosis? Accessory zone of Lissauer absolutely intact.

In these two cases the histological nature of the lesions was identical with that which we meet with in tabes. The atrophied nerve tubes are scattered in a very close meshwork of extremely fine connective fibrils crossing in all directions. Nowhere do we find ectasis of vessels, nor thickenings of the walls of vessels which penetrate from the pia mater into the cord. The diffuse general thickening of the pia mater is but slightly pronounced, and is not at all more marked at the periphery of the cord at the places affected by sclerosis. Contrary to what Friedreich thought, there cannot then be any question here of a propagation of the inflammatory process of posterior lepto-meningitis to the lateral columns.

Rütimeyer has also found some degenerated nervous fibres in the median and in the sciatic, but the degeneration of these nerves was, to sum up, very slight. On the other hand the posterior roots were everywhere degenerated, least so in the cervical, most in the lower dorsal and lumbar regions.

Primary, combined, system sclerosis was very distinct in the columns of Goll, of Burdach, in the crossed pyramidal and the direct cerebellar tracts. The lesion of these diverse systems could be followed from above downwards in the spinal cord from the highest cervical to the sacral region. We refer to the important work of Rütimeyer for the detailed description of these lesions. We shall, however, remark that in the two cases the posterior radicular regions of Westphal, the integrity of which is necessary for the existence of the tendon reflexes, were completely degenerated in the whole lumbar cord. Moreover, the columns of Clarke, and their ganglion cells in the grey substance, were affected with increasing intensity from the upper dorsal to the lumbar region.

Among the portions which have remained intact we must

quote the direct pyramidal tracts, and the "antero-external zones" of the posterior columns, and especially the marginal zone of Lissauer,† which has always been found hitherto much degenerated in ordinary tabes.‡ If this degeneration is constant in Duchenne's ataxy and is regularly wanting, as it seems to be up till now in Friedreich's ataxy, this will be a new anatomical character important to differentiate between these two diseases. (See Lissauer's diagram of the lumbar posterior cornu in BRAIN, October, 1888, p. 329.)

To sum up, after the results of the different autopsies to which we have just referred, we may conclude that from the anatomical point of view, Friedreich's disease is clearly distinguished from tabes and from the other combined sclerosses.

1st. It is distinguished from tabes by the primary combined system, degeneration of the direct cerebellar, and of the crossed pyramidal tracts, by the pronounced atrophy of the cells and of the fine fibres of the columns of Clarke, whilst the marginal zone of Lissauer always remains intact. In tabes, this zone is, on the contrary, constantly degenerated, whilst the cells of the column of Clarke, the direct cerebellar, and the crossed pyramidal tracts are intact. In tabes frequent multiple degeneration of the peripheral nerves (motor and sensory nerve roots, Déjerine); in Friedreich's disease, exceptionally a few degenerated fibres in the nerve trunks (Rütimeyer). We do not, therefore, share on this point the opinion of Rütimeyer, who admits, as a symptom common to the two diseases, degenerations in different degrees of the peripheral nerves. There is certainly a considerable and characteristic difference in this respect between the two diseases. The only anatomical characters that they have in common would thus be the integrity of the grey matter of the anterior cornua, the sclerosis of the posterior

† H. LISSAUER, Beitrag zum Faserverlauf im Hinterhorn des menschlichen Rückenmarks und zum Verhalten desselben bei Tabes dorsalis. *Archiv für Psychiatrie*, Bd. XVII. p. 377.

‡ OPPENHEIM und SIEMERLING, Beiträge zur Pathologie der Tabes dorsalis und der peripherischen Nerven-Erkrankung. *Archiv f. Psych.*, Bd. XVIII., Heft. 1 and 2. W. B. HADDEN and C. S. SHERRINGTON. The Pathological Anatomy of a case of Locomotor Ataxy. BRAIN, Part XLIII., October, 1888, p. 329. "Throughout the lumbar region, Lissauer's tract is completely sclerosed."

columns (of Goll and of Burdach), especially in their middle part, with relative integrity of the antero-external bands and atrophy of the posterior roots.

2nd. Friedreich's disease is distinguished from the other combined scleroses by the regularity of the seat of its system localisation, whilst in the other combined scleroses we meet with the most varied lesions; and especially because the disease invades the structure of which is congenitally incomplete. In all the cases of Friedreich's disease which have been examined *post mortem*, the authors have been surprised by the smallness of the cord and of the bulbs which have always been found slender and atrophied, especially in their posterior part, as well as by the smallness of the nervous elements of these organs, cells and fibres, as seen by the microscope.

Friedreich's disease, which forms an absolutely distinct clinical group, presents, therefore, also anatomo-pathological characters, which allow us to class it apart in spinal diseases. It is a combined primary system sclerosis of the spinal cord, in which several systems of fibres have been affected from birth or during infancy, with an arrest of development (posterior columns, pyramidal, and direct cerebellar tracts), and degenerate before having attained their full growth.

Prognosis and Treatment.

Two things must be distinguished in prognosis; as regards the cause of the disease, and the gravity of the affection, *quoad vitam*. If Friedreich's disease does not directly threaten life, if the patients do not die from it, since those who are affected by it may reach a comparatively advanced age, thirty, forty years, and even more (when they are not cut off by an intercurrent affection, which is most usually the case), hereditary ataxy does not the less bear a grave prognosis, for it is progressive, in an extremely slow way, it is true, but without permitting us to hope for remissions in its fatal course. We know of no case of cure of this disease. It is true that hitherto it has only been diagnosed when it was incurable. Shall we be more

fortunate in the future, when we shall be able to recognise it from its first symptoms?

We have unfortunately but little to say about the treatment in face of such a prognosis. As prophylactic measures, in the case of predisposed children, we ought to advise good hygiene, hydrotherapy, rational gymnastics, careful education, and intelligent supervision, to prevent the child contracting fatal habits of masturbation, which would, doubtless, favour the evolution of the disease by exhausting an already feeble spinal cord. We have observed on several occasions excesses of onanism in the antecedents of those who suffered from this disease, as we have also pointed out alcoholism of the parents, of the father especially, in the family history.

As for treatment properly called, we may have recourse, without hoping too much from it to two different systems. First of all to electrotherapy. Application of continuous currents, stabile and labile, to the vertebral column, by means of large electrodes (ten to fourteen milliamperes, three times a week). The patient generally feels well after the applications of electricity. He feels stronger, more active, less weak, and less uncertain on his legs. By continuing this treatment for several years, with regular intervals, we may, perhaps, obtain a decided delay in the progress of the disease. This is at least the impression which I have derived from the treatment, to which I have submitted the patient whose case has been related above. He has come to have the current applied for three years, omitting the applications two or three times each year, and he has told me that he found benefit from them. "I should not come so regularly," said he to me, "if I did not experience any benefit."

The second method is that of suspension, recommended by M. Charcot. They have obtained at the Salpêtrière some good results in this disease. I have suspended my patient more than a hundred times with a certain success. Speech has improved; the general condition has derived good from it. The patient has often found benefit from it, but the local symptoms of ataxy of the extremities and of weakness

of the legs have not been at all modified. The club-foot, the scoliosis, the nystagmus, the abolition of knee reflexes, the choreiform instability, all these exist after as before the treatment by suspension. Would more have been gained by employing Sayre's jacket, as M. Gilles de la Tourette proposed? We do not think so. But it is evident that we ought to try everything to combat a disease against which we do not really possess any efficacious means of treatment.

Appendix.

We must add to the cases which do not belong to Friedreich's disease that of J. Botkin.(63) By the courtesy of Prof. Dragomanoff, who wished me to translate it, I am able to give here a résumé of this case. Here is, first of all, the exact title of the article.

Jac. Botkin. Sloutchai Friedreich-oviboliezni. (A case of Friedreich's disease, *Medical Review*, Moscow, 1885, No. 1, p. 32-38).

Theodore Malych, age twenty-two, single, admitted November 7th, 1884, to the hospital of Yaroslaw. Mother hysterical; insane for two years after the birth of this son. Father died ten years ago, after having suffered for fifteen years from a convulsive affection, which probably had an analogy with the disease of his son, as all those who knew him affirm. The same mimicry, the same gestures, the same bearing, the same grinding of the teeth, the same movements of the eyes and of the head. The eldest brother of the patient had also in childhood convulsive attacks. His youngest brother has symptoms of "moral insanity." The patient began to walk only after his second year. He has often been ill, and in his illnesses frequently had convulsions. From eight to fifteen years of age pleurisy, scarlet fever, and typhoid fever. Each of these diseases accompanied by convulsions. Theodore Malych is timid, irascible, indolent, and obstinate. Expelled from the fifth class of the gymnasium on account of bad conduct. At sixteen years of age he enters at the special school as military surgeon's assistant. But headaches and diplopia compelled him to interrupt his

studies after seven months. After that swelling in the joints (rheumatism) for fourteen months. At the age of eighteen years (April-June, 1880), bad headaches, insomnia and melancholia. The latter lasted till December 6th. On this day on finding himself at the church of the convent of the Trinity he was taken with violent, irresistible laughter. The police took him to the hospital, where he remained four months. It is then that several symptoms of his real disease manifested themselves in the space of a few days, according to his own statement, with violent vomiting.

Present condition. Height, 167 cent.; weight, 137 Russian pounds. General pallor. Skull symmetrical. Sight normal on the right side. Left eye amaurotic. Normal reaction of pupils to light. Nystagmus. Sphincters normal. Copious salivation.

The bearing, the gestures, and the facial expression of the patient attract the attention especially. He stands erect, with the legs apart, the body thrown backwards, and leaning to the left. Head inclined backwards towards the right shoulder. Chin turned to the left and upwards, as in contracture of the sterno-mastoid. Hands crossed over the stomach. Hair thin. Apathetic expression. Jaws closed, and lips partially opened.

The voluntary movements are accompanied by a series of involuntary movements. The head is agitated by zig-zag movements, the teeth are ground. The eyes go from left to right. The arms are elevated and depressed, are flexed and extended. The hands execute movements of prehension. The body undergoes convulsive movements, forward, backward, to the left, to the right, is bent and again straightened. The feet are separated widely during walking. Trembling of the lower extremities in consequence of involuntary muscular contractions. In walking the patient sways; he is thrown from side to side. The direction of locomotion is always in a zig-zag. The legs give way sometimes and the patient falls.

The ataxy is so marked in the upper extremities that the patient has difficulty in dressing himself, and buttoning his clothes. He writes badly, and he cannot thread a needle.

When he closes his eyes, there is no increase of the ataxy, either static or dynamic. Romberg's symptom not present.

The more complicated the movement is, the more attention it demands, and the more agitated the patient is, the more do the involuntary contractions increase. The ataxy is especially pronounced when the patient walks or when he endeavours to narrate his history in a clear and distinct manner, for he never succeeds in it. It is impossible to describe the complication of the involuntary movements which are then observed. One must see them to understand to what extent they are developed.

At first sight, these movements appear disorderly, but after an attentive examination it is noticed: 1st. To each voluntary movement a definite group of involuntary movements corresponds; 2nd however numerous and varied they may be, these movements cease rapidly immediately the patient lies down horizontally and ceases efforts. The grinding of the teeth and the ocular movements form the only exception and cease only during sleep, when all the other involuntary movements also cease. Clonic contraction of the lower extremities as soon as the toes rest on the ground or on the mattress. Spasms of the muscles of the neck and of the arms. These spasms increase with the voluntary movements, and render the latter difficult, as also passive movements.

Speech is varied in tone. Far from being monotonous the sound of the voice is changed during conversation or reading; the patient passes involuntarily from one tone to another. He pronounces certain words indistinctly, does not pronounce always the end of the words. The remission of symptoms is a peculiarity of his disease. Thus sometimes he speaks much more distinctly.

Muscular enfeeblement. Dynamometer, 40 in the right, 25 in the left. The patient when lying down cannot lift his leg. If it is raised for him, he allows it to fall again.

Reflexes; plantar and cremasteric, distinct, normal; abdominal, gluteal, epigastric, scapular, but slightly marked.

Patellar reflexes and Achilles tendon reflexes *very exaggerated.* No ankle clonus.

Sensation. Rheumatic-like pains in the extremities. Vertigo. The patient localises exactly the lightest touch and the position of his limbs in space.

Spinal irritability. Slight hypho-scoliosis. Electric reactions normal.

Psychical manifestations. The patient is habitually apathetic, often excitable, and ready to laugh causelessly. Memory good. Imagination unimpaired.

In the discussion following the case, M. Botkin maintains that the presence of pareses, of contractures, and of exaggerated patellar reflexes does not exclude Friedreich's disease. He adds, however, that there exist in his patient two symptoms which have not been hitherto observed in this disease, unilateral amaurosis and grinding of the teeth. In any case, says he, even when these two symptoms would indicate a focal lesion, they cannot be regarded as due to a multilocular sclerosis. If we admit, with Féré and Erb, that Friedreich's disease is an intermediate form between disseminated sclerosis and tabes, there is nothing astonishing, concludes Botkin, in the fact that the symptoms of both lesions may be met with in this intermediate form, as has occurred in my case.

It will be sufficient to refer to the comparative table that we have given above of the differential diagnosis beeween Friedreich's disease and disseminated sclerosis to be convinced that Dr. Botkin's case does not belong at all to Friedreich's disease. We think that similar cases will not henceforth be accepted under the heading of this disease.

With regard to my provisional clinical classification of the combined scleroses, M. Dejerine writes to me :—

"Your type No. 1 (ataxic paraplegia of Gowers), and your type No. 3 (ataxospastic tabes of Grasset) are only one and the same type, clinically and anatomically, as I have established in 1884 and 1886. It is nothing else than *ataxo-paraplegic tabes of Dejerine.* I have even, unless I am mistaken, shewn you the preparations of it."

Not only has M. Dejerine shewn me numerous preparations of combined scleroses, but he has admitted me with the greatest courtesy to his admirable practice at the

Bicêtre, where he has given me permission to examine several of his patients affected with combined scleroses. I am happy to express here my sincere gratitude to him. His words have been fully opened to me, as also those of Professor Charcot at the Salpêtrière, for the observations I was desirous of making on Friedreich's disease and the combined scleroses. I owe thanks also to Professor Prevost, of Geneva, who has had the kindness to show me the preparations of the cord of the patient whose case he has published, with the autopsy, in the Archives de Physiology, 1877, and Professor Schiff, who has shewn me the cord sections which he possesses, belonging to the first patient examined *post-mortem* by Friedreich.

I must state here that I have not touched upon the study of the combined scleroses, and that the historical investigation, in particular, of this disease remains quite untouched, and has nothing to do with my clinical classification. I have not occupied myself at all in this work of the examination of the history of the combined scleroses, and I had no occasion to occupy myself with it. It would have been a digression. I have mentioned the combined scleroses only incidentally in the chapter on diagnosis, and with the sole object of showing that they could not be confounded with Friedreich's disease, when the latter presents itself under one of the forms corresponding with the four types which I have described. Now, from this point of view, it was necessary for me to separate into two distinct clinical types, ataxic paraplegia properly called and spastic ataxy. My classification has no other significance, and especially would not attempt to trench upon a historical question. If I had had to treat of this question, I venture to say that I should have recognised openly the legitimate place, and the importance which ought to be attributed, in a historical study of the combined scleroses, first of all to the case recorded by M. Prevost which dates from 1877, afterwards to the works of Westphal, Kahler and Pick, Babesiu, Raymond, Damaschino, Hopkins, Ballet and Minor, &c.; finally, especially to the memoir published in 1884 by M. Dejerine, which marks a decisive epoch in this study, and where is found for the first time, unless I am

mistaken, the complete picture of ataxo-paraplegic tabes, two years in fact before the works of Gowers and Grasset, which were printed in 1886, which is seen clearly from the bibliographical notices which I have given.

In conclusion, I shall draw attention to the work of Dr. E. Huet, *on chronic chorea*, Paris, 1888-1889, where will be found (p. 190) an interesting page devoted to the differential diagnosis of Friedreich's disease and chronic chorea, both of which are family and hereditary affections, with motor inco-ordination.

But whilst in Friedreich's disease there is choreiform instability during rest, in hereditary chorea we observe small separate, multiplied, varied and irregular movements. Romberg's sign is not present in the latter disease, nor yet nystagmus. The affections of speech differ greatly in the two diseases. The knee jerks are rather exaggerated in chorea, which is especially an affection of adult age, whilst Friedreich's disease appears, as we know in childhood and youth.

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(98) ERLICKI et RYBALKIN, Zur Frage über die combinirten Systemerkrankungen des Rückenmarkes. *Archiv für Psychiatrie*, Bd. xvii., 1886, p. 693.—Combined sclérosis of posterior fasciculi and of crossed pyramidal tracts, with complete integrity of direct cerebellar fasciculi. Ataxo-paraplegic symptoms. Interesting discussion; the authors wrongly consider the case as one of Friedreich's disease.

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(104) CHARLES L. DANA, A case of ataxic paraplegia, with autopsy. BRAIN, part xliv., January, 1889, p. 490.

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Reviews and Notices of Books.

I.—*The Anatomy of the Central Nervous Organs in Health and in Disease.* By Dr. Heinrich Obersteiner. Translated with Annotations and Additions by Alex. Hill, M.A., M.D., M.R.C.S. London: Charles Griffin & Co., 1890.

II.—*Twelve Lectures on the Structure of the Central Nervous System.* By Dr. Ludwig Edinger. Second Revised Edition, with 133 Illustrations, Translated by Wilks Hall Vittum, M.D. Edited by C. Eugene Riggs, A.M., M.D. Philadelphia and London: F. A. Davis, 1890.

I.

The original of this work was reviewed in the number of this journal for April, 1888, and the hope was then expressed that it would be translated into English. This has now been done by Dr. Alexander Hill, who has also made various annotations and additions to the original work. The translator has also added a chapter in the appendix on his theory of the rotation of the great brain—a full account of which was published in 1885 at Cambridge under the title of "The Plan of the Central Nervous System," and also a valuable glossarial index, besides inserting several fresh figures. Further, Professor Obersteiner has added much new matter and made numerous alterations so that, as the translator states in his preface, the present volume may fully claim to have the value of a new edition.

As the different sections of the work were described in detail in the review of the German edition it will here suffice to discuss the various annotations which have been made by the translator, and especially the appendix and the index for which he is responsible.

In the first section on methods, the process, first described by Barrett, of imbedding in celloidin in the usual way and afterwards taking out the spirit by water and then cutting the mass in the frozen state is recommended. In Section II. on Morphology,

Professor Hill's recent researches on the development of the nervous system from the epiblast, and the division of the cells into nemoblasts and spongio-blasts (supporting cells) is detailed.

In Section III. on the Nervous Constituents, the translator's theory with regard to "granules" is given, and a parallel is drawn between the granules of the "inner nuclear layer" of the retina and those of the olfactory bulb, and he looks upon them as the nucleus of a bi-polar cell with non-medullated processes.

In Section IV., the Histology of the Spinal Cord, is enriched by a short account of Gaskell's well-known researches on the Sympathetic System; and in the same section the question as to whether the posterior sensory roots of the cord are connected with cells or with a plexus is discussed, and here Dr. Hill takes the latter view, while the probability of the former being correct is maintained by the author. Further on, Sherrington's researches on counting the number of degenerated fibres and recording their position in the cord after cortical destruction, are mentioned. In the description of the corpus callosum on p. 347, it is stated by Professor Obersteiner that its fibres pass into the external capsule: this assertion the writer has not been able to confirm in the quadrumana, as here the fibres of the corpus callosum pass across those of the external capsule on their way to the operculum.

Besides the additions above noted, the English edition contains in the appendix a chapter by the translator on his theory of the rotation of the Great Brain, which he had previously propounded in his thesis on the Plan of the Central Nervous System in 1885. In this rotation the author considers that a loop or rink is formed, bringing the part which was at first in front on to the under surface of the back, so that the foramen of Monro, which in the embryo opens into the posterior part of its ventricle, in the adult opens into the front of the ventricle; he further states that this alteration in the position of the Great Brain was the almost necessary deduction from certain conclusions as to the connections of the olfactory tract with the anterior end of the optic thalamus, viâ the fimbria, fornix, descending pillar of the fornix, corpus mamillare and bundle of *Vicq d' Azyr*. As evidences of this connection, it is stated that in the hardened brain of the ox, the fornix, pyriform lobe, and olfactory bulb can be broken off in one piece, and that the fascia dentata is probably a continuation backwards of the glomerular layer of the olfactory bulb. As this connection is held as strong evidence to prove the rotation, it would be just advisable to consider whether it really

exists. The mere fact of hardened portions of the brain breaking off in one piece can hardly be taken as proof of functional connection, and as far as the writer has been able to ascertain microscopically, no definite connection can be made out—at any rate in the quadrumana—that the olfactory tract is in connection with the fascia dentata. It can be seen in microscopical sagittal sections that although the pyriform lobule is situated immediately in front of the cornu ammonis and the fascia dentata, there is no connection between them; they are separated by the anterior end of the descending cornu of the lateral ventricle and by the fibres of the fimbria or alvens, and no fibres can be made out connected with the two bodies; and the same thing can be made out in the brains of rodentia and carnivora. Until, therefore, further evidence is forthcoming it can hardly be laid down that the olfactory tract passes into the fascia dentata. The pyriform lobule has sometimes been described as part of the uncinate convolutions, no doubt because the cortex on the surface is more or less continuous—though on page 276 of this work it is stated that in many animals the lobule is separated from the rest of the hemisphere by the scissura limbica—but on cutting through this cortex the separation can readily be seen.

With regard to the rotation of the brain, it is assumed that the anterior end of the temporal lobe is fixed to the ethmoid bone by the olfactory bulb and tract, and that the rest of the brain rotates forwards on its transverse axes. It is difficult to understand how this slender nerve can fix the temporal lobe so as to allow sufficient traction to be exerted to produce the fan-like arrangement of the fissures in the Island of Reil, as stated in the text. Also if the temporal lobe is the fixed point, it is difficult to explain the length of the olfactory tracts and the distance of the apex of the temporal lobe from the ethmoid bone in carnivora and rodentia, for the fixation must be assumed to take place in all the mammalia. One would like to know whether the appearances cannot be explained by the flexures which take place in the growth of the brain without calling in the aid of a theory of rotation. In the lowest mammalia, such as the kangaroo, the frontal region is slightly developed and the temporal region is very far back, whilst the cornu ammonis and fascia dentata extend so far forward as almost to take the place of the gyrus fornicatus; but as we ascend the animal scale the frontal region is pushed forwards and the temporal region downwards and forwards, so that in the quadrumana and man owing to the high development of the so-called motor region, the tip of the temporal lobe comes

very near to the ethmoid, and the cornu ammonis does not make its appearance till the hinder end of the splenium is reached.

There is one addition to the English edition, for which our special thanks are due to Dr. Hill, and that is the Glossarial Index at the end of the book. He has taken great trouble to give an index of the German names used in the translation with the Latin and English names, and in many cases the French equivalents.

It would not be out of place here to raise a protest against the employment of a different word in every different language for the various parts of the brain, and we would urge that all neurologists should use the language common to all, viz.:—Latin. For instance, the claustrum is called *Vormauer* in German and *avant-mur* in French; and when we come to the *Hirnschenkel-schlinge* it takes the student of other nations a great deal of trouble to find out that it is the ansa peduncularis. Other words as the äusserer Kniehöcker (*corpus geniculatum externum*), Strickkörper (*corpus restiforme*) require the student to have a medical dictionary. In these days of international reading surely the Latin equivalent would save a great deal of unnecessary trouble, which Dr. Hill has here made an important step in alleviating. It is some satisfaction to feel that English neurologists have not erred in a similar manner, or we might have such words as the "front-wall," the "brain-shank-loop," the "outer knee-hump," and the "rope-body," as equivalents for the above words.

We must congratulate Dr. Hill on his excellent translation,—as it makes all the difference in the world to the reader whether a good English style is pursued or only a mere translation is offered, in which the foreign construction can be everywhere detected—and on the completion of a task which will bring Professor Obersteiner's valuable work within the reach of all English students of neurology.

II.

A Review by Dr. James Anderson, of the first German Edition of Dr. Edinger's Lectures has already appeared in *Brain* (Jan. 1886). A second edition was published in 1889, which has now been translated into English, and edited by the above-named authors.

Dr. Edinger states in the preface to his second edition that it

has undergone many changes, and has been added to in some respects. The chapters on histology and histogenesis have been entirely re-written, as also the sections on the oculo-motor, the acoustic, and the fibres of the deep marrow. The recent discoveries on the course of the tracts in the posterior tracts has had the effect of simplifying the description of that part. Dr. Edinger further states that he has embodied a treatise of the comparative anatomy of the nervous system based on his personal investigation.

It will thus be seen that the present presents considerable improvements upon, whilst it maintains throughout the excellent character of, the first edition. The illustrations which are particularly clear and original, have been increased from 120 to 133, and add very much to the utility of the work. The chapter and the illustrations on the subthalamic region are exceptionally good, and demonstrate the intimate knowledge which the author possesses of this most intricate part of the brain.

The English translation is most satisfactory, and we have to thank Drs. Vittum and Riggs for having made this important work of Dr. Edinger's accessible to English-speaking students of Neurology.

A. Beevor, M.D.

Sanity and Insanity. By Charles Mercier, M.B. London: Walter Scott, 1890, pp. 395.

This work treats its subject on lines different from those usually adopted. It is not intended at all exclusively for medical men and it presumes no previous knowledge of the nervous system or of the mind. It is as far as possible free from technicalities, and is written with a fluency of style and a profusion of illustrations and analogies, very unusual in medical literature.

Its main distinction, however, from any other work on the subject, so far as we are aware lies in its way of looking at Insanity, its analysis and classification of the forms of insanity. Hitherto the classifications of insanity have been what is usually termed "artificial;" that is, they have not been based on any broad fundamental relations, but on some more or less superficial point. These classifications correspond to the botanical classification based on the number and arrangement of stamens

and pistils. Suicidal maniacs are a group of insane people, and such a grouping is for practical purposes a very useful one, but it connotes very little more than the tendency to suicide, and for purposes of study of Insanity it is almost valueless. The old botanical classification is still valuable as an index to plants, but there is no suggestiveness in it. It helps us no farther forward. Similarly the old classifications of insanity are useful still for example for asylum purposes, but they do not help us to understand Insanity. The classification attempted by Dr. Mercier is a "natural" classification. It is based on the views of Dr. Hughlings Jackson regarding Insanity or rather regarding Insanities, that they are "dissolutions" of mind, that is to say, reversals of evolution. Dr. Mercier, with a knowledge of the views Herbert Spencer and Hughlings Jackson on the one side and a direct acquaintance with Insanity on the other side, has produced a work of real originality; a work which may not do much to help the so-called "practical man," but will do much to help those who desire some clue in the labyrinth of mental disorders, as well as some principles to guide them in dealing with those mentally disordered.

The first two chapters are devoted to an exposition of the nervous system, and the third to an exposition of mind. Medical men, as well as others, are sometimes singularly and even complacently ignorant of elementary psychology, and in consequence of this ignorance are ready to accept, and even to present mere verbal quibbles as true explanations of psychical phenomena. Careful perusal of these chapters would clear away, at least, some of the mist that surrounds the subject. The fourth chapter discusses "what Insanity is," and the last ten chapters are divided equally between the Causes and the Forms of Insanity. We have endeavoured to indicate the *differentia* of this work, and it would be useless to discuss these chapters in detail. Suffice it to say that they show breadth of grasp and knowledge of detail, not often combined; that they are suggestive more than exhaustive, and what for the medical as well as the lay reader is a matter of no small consequence, their interest never flags. Perhaps on this last point we might venture to say that if Dr. Mercier would discard an occasional flippancy from his writing, he would not weaken the vigor of his style, and would remove an element that is occasionally objectionable.

The work is, as we have said, intended for lay as well as medical readers, and we can recommend it heartily to both.

Such knowledge as it conveys regarding both Sanity and Insanity should be in the possession of every well-educated man, and of every well-educated woman also. When men and women know more than they do of both their mind and their body, the world will be the better and not the worse for it.

JAMES ANDERSON, M.D.

De l'Acromégalie, Maladie de P. Marie. Par SOUZA-LEITE, J.D. Paris: Lecrosnié et Babé.

THIS work aims at giving a full account of the present knowledge concerning the disease to which the name of "Acromégalie" was given by the distinguished clinician who first described it. Dr. Souza-Leite has brought together and analysed with the greatest care all the cases hitherto published. The chapters on the etiology, symptoms and treatment of the disease contain little more than is to be found in the paper on acromegaly published in BRAIN (July, 1889).

P. Marie stated then that he regretted his inability to give a full account of the anatomo-pathological characters of acromegaly, as he had performed one autopsy only. He had found in that examination that the hypertrophic process affected the spongy tissues of the bones (short bones, flat bones, epiphyses) more especially, there being at the same time hypertrophy of the vertebræ, sternum and clavicles. He stated that hypertrophy of the pituitary body, with enormous dilatation of the sella turcica, persistence of the thymus, and finally hypertrophy of the cord and ganglia of the sympathetic system, were the chief pathological appearances found at the *post-mortem* examination of acromegalic patients. Dr. Souza-Leite mentions seven *post-mortem* examinations, and, although some of these autopsies are incomplete, yet for the most part they establish on a firmer basis the accuracy of P. Marie's statements.

In the seventh chapter the author discusses the diagnosis of acromegaly from a new disease, to which P. Marie has given the name of *ostéo-arthropatie hypertrophiante pneumique*. This is by far the most important chapter in the whole book, and it will interest the readers of BRAIN if the author be quoted almost

verbatim. Let it be stated beforehand, however, that under the name of ostéo-arthropatie hypertrophiante pneumique is meant a peculiar deformity of the bones occurring in patients afflicted with some lung affection. According to the same author three cases have been mistaken for acromegaly (W. Erb, Ewald, O. Fraentzel, Gourand, R. Saundby), and for osteitis deformans (L. Elliott). The following lines occur in a lecture published by P. Marie in the *Bulletin Médical* (December, 1889):—

"The hands in this disease (pneumic hypertrophie osteo-arthritis) are truly enormous. I might even say that their size is even more astounding than that of the hands of acromegalic patients, but whereas the hands of the latter patients are merely thick and stumpy, those of patients suffering from 'pneumic hypertrophie ostéo-arthritis' give the observer the impression of something never seen before. They are not only thick, but deformed also.

"The three segments of the hand, namely, the fingers, carpo-metacarpal region and wrist must be studied separately. Of these three segments the fingers suffer most. Sometimes they are simply a little longer, though always distinctly thicker, than normal. The circumference of the first phalanx of the radius may measure 5 cm., 7, or 10 cms. for instance. These dimensions exceed those of patients suffering from acromegaly by one centimetre. The shape of the fingers in pneumic hypertrophic ostéo-arthritis is also peculiar, in so far that the last phalanx is greatly hypertrophied, and becomes so bulbous that it is often the thickest of the three. The *fingers* therefore resemble drumsticks, the thumb being like a bell-hanger. This is never the case in acromegaly, for, in the latter disease, each of the digital segments is proportionately increased. The nails covering the extremity of the deformed finger are deformed likewise. They are considerably widened (2 cms. and more for the medius), and are longer and in-curved (Hippocratic aspect), so that the extremity of the thumb of our patient, for instance, when looked at sideways, exactly resembles the head and curved beak of a parrot. [Nothing like this is ever seen in acromegalic patients whose flattened nails appear, on the contrary, to be too small for the thick phalanges which they protect.]

"Attention must also be drawn to the very noticeable longitudinal striation of nails, to their marked tendency to split, and to the formation of longitudinal fissures. Their peripheral extremities are not infrequently of a well-marked and vivid rose colour. In three cases it was also noticed that the last phalanx could be *hyperextended* with abnormal ease.

"So much for the fingers. The case is different with the *carpo-metacarpal part of the hand*, for its size and shape do not greatly differ from those of a normal hand, except that the heads of the metacarpal bones are slightly enlarged, so that there is a singular contrast between the size of that region and that of the fingers. . . . . In cases of acromegaly, on the contrary, the metacarpo-phalangeal region of the hand is enormous; I might even say that the increase in size of the metacarpo-phalangeal part of the hand is relatively more marked than that of the fingers; and this deformity gives to the hand the aspect of a 'battoir' [the spade-like hand of English authors].

"Let us now study the third segment, the *wrist*. In all cases hitherto published the widening of this region was noticed, and this condition is very noticeable on the various pictures appended to the notes of the cases. The inferior extremity of both bones of the fore-arm suddenly becomes much thicker and forms an enormous projection on the under-surface of the hand, the increase in size taking place in an antero-lateral as well as a transverse direction. The inferior part of the fore-arm thus becomes larger than the middle part, larger even than the upper part situated just above the elbow.

"Notice also that the wrists are markedly deformed, and every unbiassed person on seeing one of these wrists for the first time, would certainly give to the disease the name of arthritis deformans. Nothing like this is ever present in acromegaly, for although the wrists of some patients are thicker than those of a normal individual, the increase is quite proportionate to the size of the upper limb, and there is at this spot no projection, no sudden widening—in one word, no deformity. This widening also is never so marked. . . . .

"If this be the aspect presented by the hands of a patient suffering from pneumic hypertrophic arthritis, that of the feet is exactly similar. If the latter be also divided into three segments (toes, tarso-metatarsal region, malleolar region), we see that the toes, like the fingers, are not only somewhat lengthened, but markedly broadened, and that this process is more specially apparent in the last phalanx. They resemble a bell-clapper even more than the fingers do, the nails being enormous and curved in. The increase in size of the second segment (tarso-metatarsal region), like the increase in the corresponding region in the hand, is less marked than in other parts of the foot, although it is evident that its dimensions are larger than usual, and that the greatest increase takes place in the heads of the metatarsal bones.

"The size of the third segment of the foot (malleolar region), like the wrist, is enormously increased in all directions, the transverse bi-malleolar diameter is not less than 112mm., and this region, like the other segments, is evidently deformed. The lower part of the leg is therefore distinctly larger than the middle part; hence many authors have given to this deformity the name of 'elephant's foot.'

"This increase is due doubtless to the augmented volume of the *bones*. . . . . In acromegaly, on the other hand, the soft parts themselves, like the bones, undergo hypertrophy, and in the latter disease, therefore, we no longer deal with a hypertrophy *en masse* of all the tissues of a region. The diaphyses, more especially the articular extremities of all the long bones, are notably increased in size; but this hypertrophy does not appear to be quite symmetrical in the two bones of the segment of a limb. The long bones of the distal segments of the limbs (ulna, radius, tibia, fibula) are relatively more hypertrophied than those of proximal parts of the limbs (humerus, femur). The elbows and knees are also larger than natural, the patellæ especially showing a distinct increase. It is possible also that the kyphosis beforementioned is dependent, to a certain extent, on lesions of the intervertebral articulations.

"The patients are particularly clumsy with their hands, their elbows are in a state of more or less permanent flexion, whilst complete extension is impossible. The same remark applies to the knees also. I cannot say whether anything resembling this condition is to be noticed in the coxo-femoral articulations, but in our patient at any rate the movements of the shoulders were certainly impaired."

The last part of Dr. Souza Leite's work is a complete report of all cases of acromegaly hitherto published.

The book is profusely illustrated with 102 woodcuts, many of which are original.

M. Armand Ruffer.

Small Photographic Atlas of the Nervous System: The Brain. By Dr. J. Luys. Paris: F. B. Baillière, 1888.

This atlas is but a compendium of his large work, *Iconographie Photographique des centres nerveux*, in which the author manifested notable divergences from the generally received

opinions. Dr. Luys views with disfavour the classic description of the so-called sensory bundle, in its course through the bulb, from the posterior columns of the spinal cord to the cortex. How long, he says, can an *à priori* statement, made in the name of physiology, serve for an anatomical classification, and give a name to any nerve bundle whatever?

This criticism does not appear to us to be well founded, for anatomy cannot be considered as a science in itself, but is only one of the constituents of the whole science of the living being, which includes both morphology and the study of bodily functions. Moreover, even in descriptive anatomy we see muscles constantly receiving names derived from the part they play in the physiology of movements. Faithful to his principles, Dr. Luys gives this purely anatomical definition of the brain:—"The *ensemble* of convolutions of each lobe, connected from one side to the other (commissural fibres), and connected to the central thalamo-striated and to the sub-thalamic nuclei (white fibres: cortico-thalamic, cortico-striated, cortico-subthalamic)."

A view to which the author attaches chief importance refers to the anatomical and physiological relations between the central nuclei and the cortex. The numerous photographic illustrations which make up the second part of this Atlas (the first part of which is merely an anatomical *résumé*), and are good from the purely photographic point of view, serve the author as a basis to establish his peculiar views concerning the origin and distribution of the cerebral radiating fibres. It seems to us difficult, however, that macroscopic sections can clearly show the passage of fibres which have such varied directions, and we cannot help wondering at his reliance on this one mode of investigation. For, on the other hand, we see that Obersteiner in his classical work enumerates with details at least five methods of research, elaborated by such men as Stilling, Flechsig, Türk, Meynert, Gerlach, Gudden and Weigert.

Among the various statements of the author which appear to us difficult of admission, we find that in the brain of men of very powerful intellect Dr. Luys has noted a very large development of the convolutions in the ascending parietal region. He also reports that in cases of chronic hallucinations the para-central lobule undergoes morphological change which can be seen by the naked eye. He attributes to the region of the cuneus the property of receiving acoustic impressions. Further, the grey substance of the central regions appears to be a favourite subject of the author's who has discovered a nucleus, an accessory branch of

the red nucleus of Stilling, called by Forel Corpus Luysii. In his opinion the nucleus of Stilling is a region which is an essentially motor and excitable region; and the optic thalamus contains nuclei to which he attributes special physiological properties—olfactory, visual, &c. But one of the reasons given by the author in favour of his point of view appears rather contradictory, for he says that the white fibres of the corona radiata, which he assumes to be connected with his visual nucleus, are distributed to the antero-lateral convolutions. Dr. Luys thereby implicates that optical impressions are produced in these portions of the cortex; whereas the cortical centre for vision is usually localised in totally different parts of the brain surface, notably in the occipital lobes. The functions of these latter Dr. Luys makes out to be auditory, on the ground that he thinks he has traced into them radiating fibres from the acoustic nucleus he believes to exist in the thalamus. Finally, he ascribes trophic functions to the central thalamic region, which he describes as connected with olfactive and optic fibres.

The optic thalamus would thus be a receptive and elaborative centre for sensory impulses from the periphery on their way to the cortex. He also attributes a peculiar structure to the corpus striatum, which he describes as containing, besides its numerous polygonal or oval-shaped cells, small yellowish corpuscles infinitely ramified which appear to proceed from cerebellar elements. The yellow nucleus which he afterwards describes, the innermost portion of the lenticular nucleus, would thus bear a trophic relationship to the lobes of the cerebellum.

Again, according to Dr. Luys, the so-called converging fibres, which are in connection with the central nuclei, begin in the middle of the intercellular network of the grey cortex, and consequently are not in direct communication with the nerve cells; and the bulk of the fibres composing the internal capsule does not unite directly the cortex with the medulla, but ends in the optic thalamus. The corona radiata would thus be composed of fibres arising from the central nuclei, the corpus striatum being motor, the optic thalamus sensitive and sensorial. These, in brief, are the chief peculiarities of Dr. Luys' teachings. We see that if the opinions put forth in this work deserve to be characterised as original, they are far from being in consonance with the statements of recent authors, such as Obersteiner, Edinger, and other recognized authorities in nerve anatomy.

P. Chaslin, M.D. (Paris).

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By ALEX HILL, M.D., Master of Downing College, Cambridge.[1]

ANATOMY TEXT-BOOKS.

1. Manuale di anatomia degli organi nervosi centrali dell' uomo. MINGAZZINI (*Rome*, 1889).
2. The Anatomy of the Central Nervous Organs in Health and in Disease. OBERSTEINER AND HILL (*London, Griffin & Co.*, 1890, pp. 432, figs. 198).
3. Twelve Lectures on the Structure of the Central Nervous System. EDINGER (*translated from the second edition by* VITTUM AND RIGGS.) *Davis, Philadelphia and London*, 1890, pp. 230, figs. 133.
4. Report upon the Progress of the Anatomy of the Central Nervous System during the year 1889. EDINGER (*Schmidt's Jahrbücher*, ccxxviii., p. 73, 1890.)

ANATOMY.

5. Two Chinese Brains. DERKUM (*J. of Nervous and Mental Disease*, xiv., p. 421, 1889).

TOPOGRAPHY.

6. The Topographical Relations of the Brain to the Skull-case. MÜLLER (*Bern*, 1889, pp. 60, plates ii., figs. 8).

COMPARATIVE ANATOMY.

7. The Central Nervous System of the Cetacea. KÜCKENTHAL AND ZIEHEN (*Denksch. der Med.-Naturw. Gesellschaft zu Jena*, iii., pp. 80-200, plates iv.-xii., with tracings in outline of the life-size drawings).

[1] To my great regret I have been unable to find time to make these abstracts complete to the end of the year 1890. I shall hope to fill up the gaps in the next number of BRAIN.—A. H.

Morphology.

8. The Origin of Vertebrates from Arachnids. Patten (*Q. J. Micros. Anat.*, Aug., 1890, pp. 317-379, plates xxiii. and xxiv.)

This paper contains an elaborate description of the nervous system of the scorpion. The author contends that its cephalothoracic neuromeres, nerves, sense organs and mesoblastic somites, present, in a general way, not only the same specialisation and the same numerical arrangement in groups, but also the same difference as a whole from the body-segments as do the corresponding parts in the vertebrate head.

9. On the Origin of Vertebrates from a Crustacean-like Ancestor. Gaskell (*Q. J. Micros. Science*, cxxiii., August, 1890, pp. 379-445, pl. xxv.-xxviii.)

Several morphologists have been led to the conclusion, from a study of the vertebrate brain, that the anterior end of the alimentary canal must, in vertebrate ancestors, have pierced the floor of the third ventricle. Gaskell goes further, and says that the cavity of the central nervous system is the primitive gut, the ventricles of the brain the primitive stomach. Hence he traces the vertebrate limb of the animal tree back to a meeting point with invertebrate limbs in a proto-arthropod, not, as most morphologists suppose, in a proto-annulose animal. (*For an account of his theory see* Brain, *July*, 1889, *p.* 1.) The present paper sets forth the evidence obtained from a study of the central nervous system of young lampreys (the ammocoetes of Petromyzon Planeri). In these animals the ventricular cavity is large; its roof formed by a much folded membrane. If the membrane were unfolded a large cavity similar to a crustacean stomach would be produced. The resemblance of the infundibulum to the œsophagus of the crustacean, compressed from above downwards, is more marked than in the sheep (see previous paper). Traces are also visible of another diverticulum from the back of the third ventricle, compressed from side to side, the conus postcommissuralis (? homologous with the ganglion interpeduncular); this Gaskell regards as the altered duct of the crustacean liver. It ends in a cellular mass which invests the greater part of the brain, and consists of cells, like liver-cells, separated from one another by pigment. The pigment, Gaskell thinks, indicates the former presence of abundant inter-cellular blood-vessels. The ganglia habenulæ are large, the right being the larger. They are connected respectively with the large dorsal and small ventral pineal eyes,

and a large right and small left Meynert's bundle, and represent the optic ganglia of the pair of median eyes of the crustacean-like ancestor. The pineal eye is of the invertebrate type. The portion of the brain-case which covers it corresponds to the cuticular lens of invertebrates.

The brain is made up of a lining epithelium of fattily-degenerated columnar cells, in places ciliated; substantia gelatinosa centralis containing the tail-like processes of columnar cells, and nerve matter. The raphe is formed by the fusion of apposed layers of degenerated columnar cells. The prechordal and epichordal portions of the central nervous system (parts in front of and behind the infundibulum) agree, in minute structure, with the supra- and infra-œsophageal ganglia of the Crustacea, with which they respectively correspond. The chief difference between them depends upon the much greater number of small nerve cells, grouped in masses, in the prechordal (supra-œsophageal) than in the epichordal (infra-œsophageal) portion.

10. Address on the Origin of the Vertebrate Nervous System. GASKELL (*Brit. Med. J.*, No. 1563, pp. 1341-1345, figs. 1-10, Dec. 13, 1890).

EMBRYOLOGY.

11. Development of the Human fore-brain from the end of the first to the beginning of the third month. HIS (*Abhandl. der math.-phys. Classe der K. Sächsischen Gesellsch. der Wissensch.*, vol. xv., No. viii., pp. 675-736, figs. and plate, Nov., 1889, *Leipsic, Hirzel*).

12. The Development of the Ganglionic System in the Fowl. GOLOWINE (*Anat. Anz. V.*, No. 4).

13. A Stage in the Growth of the Primate Brain. CUNNINGHAM (*Brit. Med. J.*, Sept. 20, 1890, p. 683).

The transitory infoldings of the thin cerebral wall which appear towards the end of the second month explained as due to a want of harmony in growth between the brain case and the brain, and especially to the tendency on the part of the brain to grow backwards into an occipital lobe.

14. Address on Cerebral Anatomy. CUNNINGHAM (*Brit. Med. J.*, Aug. 2, 1890, p. 277).

The morphological value of the fissures and convolutions. Difficulty in harmonizing ontogeny and phylogeny. Convolutions, homoplastic, if not homogenetic. The transitory fissures of the

third month bear no relation to the fissures of the adult brain, and are probably peculiar to the primate brain. The early appearance of annectant gyri a characteristic human feature. Hence in Man some fissures, the Rolandic for example, begin their existence as separate pieces, which extend towards one another until they meet over the deep annectant gyri, or fail to meet, as the case may be. Critical account of the different mechanical explanations of the formation of convolutions, and conclusion that they cannot be attributed to the influence of external forces, but are due to such internal tendencies to growth as determine the form of other physiologically distinct organs.

Physiology.

15. The Function of the Central Nervous System in Invertebrate Animals. Steiner (*Sitz. d. K. P. Akad zu Berlin*, 1890, ii., p. 39).

A long series of observations on the functions of the several ganglia in various classes of invertebrates.

16. Voluntary and Reflex Muscular Contraction. Haycraft (*J. of Physiology*, xi., 4 and 5, pp. 352-369, pl. xii., July, 1890).

Physiology: Comparative.

17. The Comparative Physiology of the Great Brain. Schrader (*Deutsche Med. Wochenschrift*, No. 15, 1890).

Investigation of the function of the bird's brain, using for experiments the owl and hawk as presenting a higher type of brain, than the dove and hen which have hitherto been the favourite subjects. The right hemisphere was removed in the owl, which became lame with its left foot and blind with the left eye, although flight was not affected. In the hawk both frontal lobes ("frontalhirne") were removed, and the operation resulted in complete spastic paralysis of the legs, while wings and tail acted normally. The paralysis subsequently disappeared completely. It appears from these experiments that whereas the limb muscles are not represented in the cerebral hemispheres of the dove or hen, birds of prey approach mammals so far as the cerebral representation of the hinder extremities is concerned.

Physiogeny.

18. The Effects which follow Destruction of different Parts of the Brain of New-born Animals, and the Development of the Functions of the same. Bechterew (*Medizinskoje Obosrenje*, No. 4, 1890).

The effects of destruction of a portion of the brain of a new-born animal are similar to those which follow the same operation in the adult, provided the fibres of this part are already medullated. No result is obtained if the fibres are still without medullary sheaths. Destruction of the motor area or section of the crus cerebri in a blind puppy are without effect, whereas section of the already-medullated restiform body produces rolling movements. The guinea pig is more highly-developed at birth, and injury to its brain at this time produces the same results as in the adult animal. Bechterew investigates the successional development of sensory and motor functions in the cortex.

Psychology.

19. The Duration of Memory-pictures. Paneth and Exner (*Centralbl. f. Physiol.*, iv., 3, pp. 81-83, 1890).

Sensory impressions give rise to "primary memory-pictures." When attention is directed to these pictures projected upon our consciousness they are fixed as secondary memories. If, owing to pre-occupation, attention is not directed to the primary memory, it rapidly fades. The paper considers the duration of this primary memory.

20. Psycho-Physiological studies in Protista. Verworn (*Jena, Gustav Fischer*, 1889. *Physiol. Centralbl.*, iv., 4, 1890, pp. 103-108).

Cerebrum: Cortex.

21. The Origin of the Convolutions of the Great Brain. Schnopfhagen (*Leipsic, Deuticke*, 1891, pp. 122, figs. 18).

Description of the white matter of the cerebrum, as investigated by de-fibering. Conclusions from the disposition of the fibres, as revealed by this method, as to the causation of convolution.

22. Intraparietal Sulcus. Cunningham (*J. Anat. and Phys.*, xxiv., pp. 135-155, 1890).

Of sixty-two hemispheres examined only four exhibited an intraparietal sulcus divided into three separate parts. The disposition usually considered as normal (ramus verticalis superior distinct from the confluent rami horizontalis et verticalis inferior) only occurred in ten. The united vertical and the horizontal rami were distinct in ten. The three parts of the sulcus were confluent in thirty-five. The lower vertical limb was confluent with the horizontal limb, the upper vertical being independent, in two. The ramus occipitalis was quite distinct from the ramus horizontalis in thirty-six, and in ten others they were partially separated by a bridging convolution. The sulcus occipitalis transversus of Ecker is regarded as belonging to the intra-parietal system, and not, therefore, homologous with the external parieto-occipital fissure of apes.

Cortex: Physiology.

23. Changes in Excitability in the Psychomotor Cortex. Landois (*Verhandl. der Medic. Vereins zu Greifswald*, 1888-1889).

24. Partial Cerebral Atrophy of Peripheral Origin. Sibut (*Paris, Baillière et Fils*, 1890, pp. 84).

A collection of recorded cases in which the cortex was found to be atrophied as the result of infantile paralysis or amputation of a limb. Conclusion: the atrophy is the more marked the younger the subject at the time of the peripheral lesion, and the longer the period which has elapsed since the lesion; it is due to functional atrophy, not degeneration; it affects the motor centre of the limb affected; the centre for the upper limb lies in the marginal (transverse) convolutions at the junction of their upper and middle thirds, and by preference in the ascending parietal convolution; the centre for the lower extremity is situate in the upper part of these same marginal convolutions and in the paracentral lobule; it usually occupies the ascending frontal. Histologically the atrophy consists in a diminution of pyramidal cells.

25. Descending Degenerations which follow lesions of the Gyrus Marginalis and Gyrus Fornicatus in Monkeys, with appendix on Degenerations which follow removal of the External Motor Cortex. France (*Phil. Trans. Roy. Soc.*, 180, B, pp. 331-354, plates 27-29, Jan. 15, 1890).

Horsley and Schäfer had found that stimulation of the gyrus

fornicatus gives rise to no muscular contractions; its removal is not followed by paralysis, but does cause deficiency in general and tactile sensibility of the opposite side of the body. France finds that removal of either the marginal or the fornicate gyrus is followed by descending degeneration of the opposite lateral pyramidal tract, traceable to the lower lumbar region. When the marginal gyrus has been removed the degeneration in the cord is confined to the portion of the tract which borders on the direct cerebellar tract. After removal of the fornicate gyrus it occupies the whole sectional area of the lateral pyramidal tract.

26. The Visual Area on the Surface of the Hemisphere. BECHTEREW (*Archiv psichiatrii, neurologii, &c.*, No. 1, 1890, *in Russian. Neur. Centralbl.*, 1890, p. 237, *abstract*).

Digest of results of various observers criticised in the light of Bechterew's own observations. He finds the area widely extended, including the whole occipital and part of parietal lobes and reaching in a coronal direction from the temporal lobe over on to the median surface. It is divisible into two great regions, of which one presides over the retina of the same side, and the other over the retina of the opposite side.

27. The Physiology of the Movements and Convulsions liberated by the Cerebral Cortex. KORANYI AND TAUSZK (*Internat., Klin. Rundschau*, No. 14, iv., 1890).

Having exposed a cerebral hemisphere in the rabbit, they painted the surface with Liebig's extractum carnis, by which means clonic contractions of the face muscles are provoked, but not general convulsions. During this state of excited activity, however, stimulation of the skin of the face provokes contraction of the right fore limb, and when stronger of the hind limb, and then of the opposite side of the body. The face is, in fact, an epileptogenous zone. To determine whether the cortex is merely a node in the reflex arc, the authors removed portions of its substance and eventually the whole hemisphere, but found that while at first the reflex convulsions were in this way inhibited, they could after a time be obtained in full force. They conclude that the convulsions are obtained through a reflex mechanism in the mid-brain.

28. The Croonian Lectures on Cerebral Localization. FERRIER (*British Medical Journal*, Nos. 1536-1541, June 7 *et seq.*, 1890, with illustrations.

29. Sight Area and Eye Movements. MUNK (*Sitz. d. K. P. Akad., Berlin*, Jan. 16, 1890). See BRAIN, part 49, 1890.

30. The first Temporal Convolutions, right and left, in a Man Deaf with the Left Ear. MANOUVRIER (*Bull. de la Soc. de Psychologie Physiologique*, 1889, p. 24).

Bertillon, the statistician, was deaf with his left ear (from ten years old) and left-handed. The right upper temporal convolution was notably smaller than the left. As is to be expected in left-handed people, Broca's convolution was better developed on the right than on the left side. A noticeable difficulty in speech which he exhibited may have been due, therefore, to his speaking with the deaf hemisphere. The optic region on the right side was considered as showing a compensatory development.

CORTEX: CONDUCTING PATHS.

31. The Central Convolutions a Central Organ for the Posterior Columns. FLECHSIG AND HÖSEL (*Neur. Centralbl.*, July 15, 1890, pp. 417-419).

Flechsig has already taught on developmental grounds that tracts of fibres connect the posterior columns with the cortex of the posterior central convolution *via* the "tegmental radiations" and fillet. Authors now describe a case in which paralysis of fifty-two years' standing (commencing in the second year) is associated with porencephalia in the region of the central convolutions. Atrophy of the left posterior central convolution, part of lobulus paracentralis, medullary substance under the upper third of the anterior central and front of upper parietal convolution is accompanied by atrophy of the left pyramidal tract and also of the fillet-layer (of the pons especially), left interolivary layer, fibræ arcuatæ internæ, and the nuclei funiculi cuneati, and to a less extent funiculi gracilis. The left red nucleus and right brachium conjunctivum cerebelli and nucleus dentatus are also small.

CORTEX: HISTOLOGY.

32. The Brain-Cortex and the Central Origin of the Nerves. MARTINOTTI (*Intern. Monatschr. f. Anat. und Physiol.*, vii., 2, p. 69).

The writer is of opinion that the course of nerve-fibres can be rendered evident by the use of Golgi's method, and that protoplasmic and nervous processes can be distinguished. Protoplasmic

processes are never in connection with the nerve network, but join neurogleia cells. Nerve cells can be distinguished (functionally) according as they are directly or indirectly connected with nerve-fibres. The former belong to motor, the latter to sensory, regions.

Corpus Callosum.

33. The Effects of Section of the Corpus Callosum. Koranyi (*Pflüger's Archiv.*, xlvii., pts. 2 and 3, 1890).

The corpus callosum was cut through in dogs. When successfully accomplished the operation gave rise to no obvious disturbance of movement. If the hemisphere was injured temporary disturbances of vision, common sensation and movement resulted; as they did also if the white matter was injured even at some distance from those portions of the cortex in which these functions are said to be localised. The results of these accidental injuries are detailed. Section of the corpus callosum may give rise to general convulsions.

34. The Brain without Corpus Callosum. Jelgersma (*Neurol. Centralbl.*, No. 6, pp. 162-167, March 15, 1890).

Pathological evidence in favour of Jelgersma's theory as to the cause of convolution in the cortex of the cerebrum and cerebellum (*cf. these abstracts*, Brain, 1889, p. 410).

Stem-Ganglia.

35. The Physiology of the Infracortical Ganglia and their connection with Epilepsy. Ziehen (*Arch. f. Psychiatrie*, xxi., 1890).

Pricking, weak faradization and section used as stimuli to the ganglia in rabbits exposed by removal of the hemispheres and fornix. Stimulation of the nucleus caudatus gave rise to such contractions as result from stimulation of the anterior part of the motor region of the cortex. No nodulus cursorius in Nothnagel's sense was found. From the nucleus lenticularis the same effects were obtained. Stimulation of the optic thalamus produced contractions on both sides of the body. Section at back of thalamus (especially across the tegment through the internal geniculate body) gave rise to running movements and cries. Movements also resulted from stimulation of the corpora quadrigemina. The author looks upon these movements as reflex; the sensory tract including the optic tract, auditory nerve, &c. The pyramidal tract was not

the motor tract for these movements. Tetanic contractions are characteristic of the infra-cortical ganglia as contrasted with the clonic spasms of cortical origin. Hence Ziehen concludes that the tonic components of the epileptic fit are liberated not from the cortex but from the stem-ganglia.

36. Effects upon the Bodily Temperature of Lesions of the Corpus Striatum and Optic Thalamus. WHITE (*J. of Physiology*, x., 1 and 2, pp. 1-25, Jan., 1890).

A variety of control-experiments performed to make sure that no cause, except the injury to the grey matter, causes rise in temperature. All precautions being taken, it was found that in rabbits the body temperature is raised on both sides of the body after unilateral injury to the corpus striatum or optic thalamus. Injury to the white matter in the neighbourhood of these grey masses produced no result.

PES PEDUNCULI.

37. The Fibre System of the Pes Pedunculi and Cortical Relations of the Corpus Geniculatum Internum. ZACHER (*Neur. Centralbl.*, July 15, 1890, p. 440).

MID-BRAIN.

38. The Origin of the deep Medullary Substance of the Region of the Corpora Quadrigemina. HELD (*Neur. Centralbl.*, Aug. 15, 1890, No. 16, pp. 481-483).

Developmental study in kittens and young rats after Flechsig's method.

MID-BRAIN: FILLET.

39. The Physiology of the Fillet (a case of Glioma of the Posterior Horn). ROSSOLYMO (*Arch. f. Psych. u. Nervenkrankh.*, xxi., 1890).

Great disturbance of sensibility; degeneration of the posterior roots, and diminution in the number of fibres in the right inter-olivary layer, pyramid, fillet, pons and crus cerebri. Goll's column unaffected. Conclusion: that the last-mentioned has no immediate connection with the posterior roots; that the fillet is made up chiefly, if not entirely, of fibres which convey skin-sensibility from the opposite side of the body.

Mid-Brain: Corpora Quadrigemina.

40. The Connections between the Posterior Tubercles and the Auditory Nerve. Flechsig (*Neurol. Centralbl.*, 4, Feb. 15, 1890, pp. 98-100).

Fresh observations, in conjunction with Held, on the brains of kittens, which (*contra* v. Monakow) confirm the author in his views as to the connection of the ramus cochlearis with the posterior tubercles *via* the anterior auditory nucleus and corpus trapezoideum. Flechsig's former observations were made on human brains. In the cat at birth the ramus vestibularis is not yet myelinated. Its development shows that the primary cochlear centre gives rise to four systems of fibres at least. Three of these take part in the formation of the corpus trapezoides, while the fourth crosses partially behind the latter in the raphe and joins it in the neighbourhood of the upper olive. The lateral fillet is connected with the ramus cochlearis by constituents of the corpus trapezoides, and also by these fibres which cross behind it. The latter come chiefly from the tuberculum acusticum, the former from the anterior nucleus.

In Man the central auditory apparatus is less developed than it is in the lower animals. Flechsig thinks it not impossible that the striæ acusticæ (which are first myelinated some time after birth) are connected with the posterior tubercles.

Mid-Brain: Optic Lobes.

41. Time Relations of Stimulation of the Optic Lobes of the Frog. Wilson (*J. of Physiology*, xi., 6, pp. 504-508, Nov. 1890).

Delay of stimulus in optic lobes determined at ·02," being exactly the same as in the case of the cerebral hemisphere. Curve of movement resembled the characteristic cerebral cortical curve. Conclusion: that the cells in the optic lobes of the frog have motor connections similar to those of the cerebral cells in the mammal.

Cerebellum: Function.

42. The Function of the Cerebellum. Gowers (*Neur. Centralbl.*, No. 7, 1890, pp. 194-5, April 1).

A condensed account of a theory of the function of the cerebellum which can hardly be still further abstracted. In outline

it is as follows:—A distinction has to be made between the central lobe of the cerebellum and its hemispheres. The former presides over co-ordination of movement, perhaps acting through the cortex of the hemispheres. The two conducting paths connected with the cerebellum (lateral cerebellar tract and posterior column) conduct upwards. These tracts are apparently ascending continuations of muscular nerves. The impulses which ascend these tracts are not, as a rule, sensible, although they govern the position of the body. Impulses also ascend from the acoustic nerve, and similarly the organ receives information as to the eye-ball movements. We do not know that a single fibre descends from the cerebellum to the spinal cord. Routes are known, on the other hand, through which an influence may be exerted directly or indirectly upon the grey matter of the optic thalamus or corpus striatum.

From these anatomical data Gowers concludes: that the middle lobe of the cerebellum is a regulating centre for such centripetal impulses as have a special relation to movement; that the cerebellum exercises a controlling influence upon the cells of the cortex cerebri, just as the motor cortex controls the centre for muscle reflex upon which the so-called tendon-phenomenon depends.

This explanation of the function of the organ serves to explain the loss of patellar reflex in tumour of the middle lobe of the cerebellum; the diminution of patellar reflex in such diseases of the cerebellar hemispheres as interrupt the route from the cerebellum: many of the phenomena of athetosis and cramps in cases of disease of the cerebellum, and the peculiar relation between the symptoms of disease of the middle lobe and those of tabes—ataxy in each case being due to disease of ascending muscle-nerves.

43. Functions of the Cerebellum. BECHTEREW (*Medizinskoje Obosrenje*, 13-14, 1890).

44. The Question of the Function of the Cerebellum. BECHTEREW (*Neurol. Centralbl.*, June 15, 1890, No. 12.)

A criticism of the theory as to the function of the cerebellum developed by Gowers in the same journal, No. 7. (See *supra*.) Bechterew particularly combats Gowers' statements as to the limited connections of the cerebellum, and the absence of descending spinal tracts.

CEREBELLUM: HISTOLOGY.

45. The Nervous Fibres of the Granular Layer of the Cerebellum and the Evolution of the Cerebellar Elements. RAMON Y CAJAL (*Intern. Monatschr. f. Anat und Physiol.*, vii., part 1).

NERVES: CRANIAL: II.

46. The Crossing of Nerve Fibres in the Chiasma Nervorum Opticorum. DELBRUCK (*Arch. f. Psychiatrie*, xxi., 1890).

The right uncrossed bundle alone was normal, and hence the subject presented a good opportunity of studying the disposition of this tract. In the nerves its fibres are collected into thick bundles, and lie on the lateral side. In the chiasm and tract they are mixed up with the crossed fibres. They are not, however, scattered uniformly over the tract, but, at first, leave the inner and under quadrant free, and farther back a continually contracting border-zone. Retrospect of the literature.

OPTIC CONNECTIONS.

47. Basal Hemianopsia. REMAK (*Neurol. Centralbl.*, 5, Mar. 1, 1890, pp. 130-136).

NERVES: CRANIAL: III.

48. The Anatomy of the Oculomotor Centres in Man. PERLIA (*v. Graefe's Archiv. fur Ophthal.*, xxxv., 4, p. 287).

Perlia divides the groups of cells from which the nerve originates into two large groups, of which the hinder is the larger. Each of these is again made up of small divisions (A), the hinder or chief group comprising: (1) hinder ventral, (2) hinder dorsal, (3) anterior ventral, (4) anterior dorsal, (5) central, (6) mediolateral (Edinger-Westphal small celled group); the anterior (B) comprises: (1) the lateral anterior nucleus, (2) the median anterior double nucleus.

Fibres from the oculomotor nuclei run into the posterior longitudinal bundle, which also contains fibres from the ansa lenticularis. A network of fine fibres in the central grey matter is also in connection with this bundle.

49. The Number and Calibre of the Nerve Fibres of the common Oculomotor Nerve in the New-born Kitten and adult Cat. SCHILLER (*Comptes rendus*, cix., 14).

Number of fibres about 3,000, and so nearly the same in the kitten and cat that Schiller concludes that there is little or no accession in extra-uterine life. The finer fibres in the new-born animal are easily overlooked.

Size: In new-born kitten, 1.5—2 μ; at one month old, 4—6 μ; at one year, 6—12 μ; at eighteen months, 6—12 μ.

Nerves: Cranial. VIII.

50. The Origin and Central Course of the Auditory Nerve in the Rabbit and Cat. Baginsky (*Virchow's Archiv*, ix., 1, p. 81).

The cochlea was destroyed in a kitten at birth, and brain examined after the animal had lived eight weeks. The posterior root, with its intercalated nerve cells, second and third layers of the tuberculum laterale, anterior auditory nucleus, corpus trapezoides, striæ medullares and superior olive were atrophied. After the crossing in the corpus trapezoides the atrophy was continued through the inferior fillet into the posterior brachium, and the ventral medullary layer of the posterior corpus quadrigeminum. In the cat the larger mesial part of the posterior auditory root goes into the anterior nucleus, the smaller lateral part into the tuberculum laterale. The outer and inner nuclei were intact. So, too, was the mesial geniculate body. (*See also these abstracts*, Brain, 1889, p. 421).

Nerves: Cranial: V.

51. Hemiatrophia Facialis and the Origin of the Trigeminal Nerve. Homén (*Neur. Centralbl.*, July 1 and 15, 1890, pp. 385-388 and 430-438).

A carcinoma of the dura mater pressed upon the Gasserian ganglion, the trigeminal nerve and its branches. The resulting atrophy of the roots of the nerve was observed microscopically. The degeneration backwards affected, chiefly, at least, the portio major in the pons, and of the several internal roots of the nerve the "ascending" was atrophied; so too was the cerebellar root. The "descending" root was less affected. No alteration was visible in the root from the substantia ferruginea. The cells were diminished in the sensory nucleus.

Certain degenerated fibres were found in the facial nerve, due apparently to atrophy of the great superficial petrosal nerve. The cornea was cloudy and ulcerated.

SENSES: SEMI-CIRCULAR CANALS.

52. Motor Disturbances after removal of the Semi-circular Canals. EWALD (*Centralbl. f. d. Med. Wissench.*, 1890, p. 114.)

After removal of the utricle, ampullæ and canals in the dove, the animal suffered from diminution in muscular power on the same side, particularly shown in the weaker movements of the wing when it attempted to escape from being held. This weakness the author attributes to the abolition of sensory impulses normally originating in the labyrinth.

MEDULLA: ARCIFORM FIBRES.

53. The Course of the Fibræ Arcuatæ Externæ Anteriores. PAL (*Stricker's Arbeiten, Vienna*, 1890).

These fibres come out from the raphe, bend round the pyramid, and enter the corpus restiforme. At the level of the upper third of the olive, the bundle coming out of the raphe gives fibres to the pyramid, only a portion of its fibres going on to invest the olive. Some of these fibres are nevertheless continued through the pyramid to the olive by a roundabout course.

SPINAL CORD: WHITE COLUMNS.

54. Number and Distribution of the Medullated Fibres in the Frog's Spinal Cord. GAULE (*Abh. der Math.-Phys. Classe d. K. sächs. Gesellsch. d. Wissensch.*, XV., 9, pp. 737-780, 10 plates, 1889).

The number of fibres in the white matter of the cord was counted at five different levels, viz.: at the junction with the medulla; in the middle of the cervical swelling; in the middle of the dorsal cord; at the beginning of the lumbar swelling, and beneath the ninth nerve. An attempt is then made to allot the fibres to peripheral nerves, using the tables drawn up by Birge for the fibres in the anterior and posterior roots. Gaule's figures tally with Birge's on the supposition that each root-fibre is connected with eight spinal fibres. He considers the bearing of this determination, that each root-fibre is in relation with a fixed number of cord-fibres, upon his view as to the position of the cell in the animal economy.

SPINAL CORD: STIMULATION.

55. Muscle-contraction following Stimulation of the Spinal Cord and the Brain with interrupted Electrical Stimuli.

HAYCRAFT (*Centralblatt f. Physiol.*, iv., 6, 1890, pp. 153-4; see also *Journal of Physiology*, vol. xi., p. 352).

SPINAL CORD: NUCLEI.

56. The Nucleus of Origin of the Nerve which innervates the M. quadriceps cruris. LEHMANN (*Dissertation, Würtzburg*, 1890).

Extirpation of the muscle in the new-born animal resulted in atrophy of the grey matter of the cord between the fifth and seventh lumbar nerves (corresponding to 2-4 lumbar in Man). The attempt to use the results for the purpose of deciding as to the true reflex nature of the "patellar reflex" was unsuccessful.

SPINAL CORD: POSTERIOR COLUMNS.

57. The effects which follow Section of the Posterior Columns of the Spinal Cord and the relation of these Columns to Equilibration. BECHTEREW (*Archiv. f. Anat. und Phys. Phys. Abth., v. and vi.*, pp. 489-505, 1890).

Author is convinced that the fibres of Goll's column originate in the cells of the posterior horn, and partly also of Clarke's column, while the shorter (segmental) fibres of Burdach's column originate almost exclusively in the cells of Clarke's column. In the experiments here recorded he cut the posterior columns in the upper cervical region in doves, rabbits and dogs. The disturbance of equilibrium was never so pronounced as is the case when the inferior cerebellar peduncles are divided. From this the author concludes that centripetal impulses which determine the action of the cerebellum as an organ of equilibration, come from other parts of the cord beside the nuclei of the posterior columns, presumably the lateral cerebellar tract.

SPINAL CORD: CONUS MEDULLARIS.

58. Disease of the Conus Medullaris and Cauda Equina of the Spinal Cord. BECHTEREW (*Wratsch*, No. 39. Sep. 27, 1890).

PYRAMIDAL TRACTS.

59. Relative Development and various Dispositions of the Pyramidal Tracts in Man and Animals, and on the

Nutrition of these Tracts. BECHTEREW (*Medizinskoje Obosrenje*, Nos. 13-14, 1890).

ANATOMY: CONDUCTING PATHS.

60. Doubled Crossing of Cerebro-spinal Conducting Paths. UNVERRICHT (*Neur. Centralbl.*, No. 16, pp. 483-487, August 15, 1890).

Stimulation of the brain of a dog between the centres for the extremities caused flexure of the spinal column to the same side. The author set himself to decide whether this is a case of homo-lateral innervation or of recrossing. The spinal cord was cut half through, either just below the skull or in the upper dorsal region. In either case the curvature to the same side as the stimulation resulted, although the spinal cord on this side of the body was cut across.

In a number of dogs the spinal cord was divided on one side and the animal observed as long, subsequently, as possible. With the paralysis of the hind foot on the same side was associated a condition of the trunk which could only be explained by supposing that the contra-lateral musculature was paralysed. If the cord had been divided on the left side the animal maintained its trunk in a curve concave towards the left.

SPINAL CORD: HISTOLOGY.

61. Outlying Nerve Cells in the Mammalian Spinal Cord. SHERRINGTON (*Proceedings of the Roy. Soc.*, Jan. 30, 1890)

A study of the nerve cells which are scattered throughout the white columns. In the anterior columns they are multipolar, and lie for the most part amongst the fibres which join the anterior commissure. In the lateral columns spindle-shaped cells are scattered about in the neighbourhood of the intermedio-lateral tract, near the processus reticulares and (especially in the lumbar cord) near the substantia Rolandi; the cells last-named appear to be connected with the fibres of the posterior roots. Cells of Clarke's column, 70μ in diameter and of broadly oval form, are found in the posterior columns, especially in Man. They are placed with their long axes in the direction of the fibres which enter Clarke's column from Burdach's column.

62. Out-lying Nerve-cells in the Mammalian Spinal Cord. SHERRINGTON (*Phil. Trans. Royal Soc.*, 181, 1890, B. pp. 33-48, pl. 3, 4).

Description of cells which are scattered in the anterior, lateral and posterior white columns. No group of cells in the lateral column which can be regarded as the homologue of the group found at the periphery of the cord in the lateral column of the alligator, &c., but a sub-pial thickening or septum which separates the lateral cerebellar from the antero-lateral ascending columns seems to indicate its situation. Cells in the radicular zone of postero-external column looked upon as belonging to Clarke's column, and suggestion that the solitary cells in the median limb of the substantia gelatinosa are also equivalent to cells of Clarke's column. Similar outlying cells on median side of cuneate nucleus. These and cuneate nucleus (*not* vagus nucleus) regarded as belonging to Clarke's column.

REFLEX ACTION.

63. The Knee-jerk after Section of the Spinal Cord. REICHERT (*Journal of Nerv. and Mental Disease*, xv., p. 71, 1890).

In twelve dogs in which the spinal cord was cut across in the cervical or upper dorsal region it was found that the most varied peripheral stimuli had no appreciable effect upon the strength of the tendon reflex. From this it is concluded that the fluctuations in extent of movement due to stimulation of other sensory nerves depends upon cerebral action.

64. Knee-jerk and its Physiological Modifications. BOWDITCH and WARREN (*J. of Physiology*, xi., 1 and 2, pp. 25-65, Jan., 1890).

REFLEX: CRIES.

65. Reflex Cries in Animals. LABORDE (*C. R. Soc. de Biologie*, 1890, pp. 83-84. Feb. 15.)

After rapid removal of the brain as far down as the pons (in guinea-pigs) reflex cries follow with perfect regularity whenever the foot is pinched.

NERVES: PERIPHERAL: SYMPATHETIC.

66. The Innervation of the Stomach. BECHTEREW and MISLAWSKI (*Neurol. Centralbl.*, 7, April 1, 1890, pp. 195-199).

67. Central and Peripheral Innervation of the Intestine. BECHTEREW and MISLAWSKI (*Arch. f. Anat. u. Phys. Phys. Abth., Supplement*, 1889).

Contractions of the gut registered by a manometer connected with a gutta-percha ball, included in the intestines of the dog. Conclusion that the normal tone, the rhythmic contractions and the slower peristaltic movements depend upon a local nerve-apparatus. The vagi chiefly innervate the small intestine and the upper segment of the large intestine. They contain more motor than inhibitory fibres. The splanchnics are chiefly, but not exclusively, inhibitory. Stimulation of the sigmoid gyrus or of the optic thalamus sometimes induced contraction, sometimes quiescence.

68. Development of the Sympathetic Nervous System in Mammals. PATERSON (*Phil. Trans. Royal Society*, vol. 181 (1890), pp. 159-186, pl. 22-30).

This monograph commences with some account of the views held as to the physiology, morphology and embryology of the sympathetic system; the two views with regard to its development (the subject upon which the author's investigations throw light) being (1) Balfour's view that the sympathetic ganglia are ectodermal in origin and arise as buds from the spinal nerves; and (2) Onodi's, that they are formed by direct proliferation of the spinal ganglia.

Paterson's investigations upon mice, rats, rabbits, and human embryos show that the sympathetic chain is of mesoblastic origin; that it arises on either side of the body as a solid unsegmented rod of cells lying in close proximity with the aorta, between this vessel and the cardinal vein, on the ventral side of the intercostal arteries. The rod reaches from the level of the cephalic border of the fore limb to the middle of the trunk (region of the stomach). It commences abruptly, is largest in front, and tapers off gradually behind. It is at first totally unconnected with the spinal cord, ganglia and nerves.

Its subsequent connection with the spinal cord is due to the extension into it of the splanchnic branches of the spinal nerves. Each splanchnic branch is connected with both anterior and posterior spinal roots. Only after this connection does it assume a segmental appearance.

A considerable column of cells can be seen in sagittal sections to slope downwards and backwards from the ventral aspect of the main cord to enter a mass of mesoblastic cells situate at the

anterior end of the embryonic kidney. The sympathetic component becomes the medulla, the other mass the cortex of the supra-renal capsule.

69. Local Paralysis of Peripheral Ganglia and connection of different Classes of Nerve Fibres with them. LANGLEY and DICKINSON (*Proc. Royal Soc.*, 7, 46, pp. 424-431, 1890).

Discovery that nicotin paralyses the cells of sympathetic ganglia, affording thus a means of distinguishing fibres which end in the cells of the ganglia from fibres which pass through the ganglia without interruption. Investigation of the relation to fibres of the cells of the superior cervical and solar ganglia.

70. Progressive Paralysis of the Different Classes of Nerve Cells in the Superior Cervical Ganglion. LANGLEY and DICKINSON (*Proc. Royal Soc.*, vol. 47, pp. 379-390, 1890).

NERVE-MUSCLE.

71. Rhythm of Muscular Response to Volitional Impulses in Man. GRIFFITHS (*J. of Physiology*, ix., pp. 39-54, pl. ii., 1890).

Griffiths agrees with other investigators, that an unweighted muscle contracts under the influence of the will at the rate of not more than ten vibrations per second. The number of interruptions is greater in a long muscle than in a short one. It increases with increased load up to a certain point, and then decreases.

BLOOD VESSELS: CIRCLE OF WILLIS.

72. Specimen, showing anomaly of the Circle of Willis. LLOYD (*Journal of Nervous and Mental Disease*, xv., p. 225, 1890).

73. The Mechanics of the Blood Supply of the Brain. GEIGEL (*Stuttgart, Enke*, 1890, pp. 45).

MEMBRANES.

74. The Termination of the Dural Sac in the Human Spinal Canal. WAGNER (*Arch. f. Anat. u. Phys. Anat. Abth.*, p. 64, 1890).

By injection it was determined that in the child the dural sac terminates, as a rule, at the level of the upper third of the third

sacral vertebra; in the adult somewhat higher. Below this it fuses with the structures which constitute the filum terminale.

Nutrition: Poisons.

75. The Action of Strychnine upon the Cerebrum. Bernatzki (*Wratsch*, Nos. 6-8, 1890).

The subcutaneous injection of strychnine in rabbits depressed the irritability of the motor centres. Washing the cortex with a watery solution of the drug has the same effect. This result is not obtained, however, so soon as in the former case, requiring thirty minutes for its development, and the author concludes that the drug acts upon the spinal cord.

Histology.

76. The Texture of the Central Nervous System of the higher Worms. Haller (*Arb. d. Zool. Inst. zu Wien*, viii., 2, pp. 175, pls. 5; also Holder, *Wien*, 1889).

In invertebrata, in addition to the "direct" origin of peripheral nerves from nerve cells, an "indirect" origin from Leydig's molecular substance has been proved. Different views are held as to the nature of the last-named substance. According to the author it varies in texture in different animals, but when best developed consists of a nervous network, supported by a coarser neurogleia-network. The paper treats especially of the phylogeny of the neurogleia-network which may merely invest the ganglion, or may form a supporting tissue for the cells, or lastly, may not only support the cells, but also form a network in the molecular substance. The direct and indirect connection of peripheral fibres is proved for all worms, also the crossed connection with the network of the colossal fibres.

In errant polychaetes the whole central nervous system is enveloped in a covering which lies immediately beneath the basal membrane (hypodermis) of the skin. This investment sends processes into the nervous tissue in which they form a network and function as connective-tissue. The meshes of the net are large where they enclose the nerve-cells, fine in the central nerve substance where they support the nerve-plexus. Neurilemma and supporting tissue (which latter is identical with the neurogleia of vertebrates) form, therefore, an organic whole. Haller divides this tissue into "perineural neurogleia-network," and central "neurogleia-network." Judging from its appearance and

re-action to stains the perineural network of the abdominal cord differs chemically from that of the brain.

Both in Lepidasthenia and Nereis the nerve-cells are for the most part pear-shaped, and apparently unipolar. The apparent unipolarity depends upon the situation of the cells outside the grey matter, through which alone they give processes; all processes may end in the grey matter, or a strong process may go into a commissure, or into a peripheral nerve. Direct connection between cells lying some distance apart was observed.

The cells are arranged in a dorso-lateral and a ventral group, between which lie the colossal cells. The large process of the colossal cell passes across into the last of the three peripheral nerves of each section; as it traverses the grey matter it gives fine branches to it on either side. The lateral colossal fibre is constructed from the nerve-network. Some of the smaller pear-shaped cells give off the fibres of peripheral nerves, others yield only branching processes for the network. A second class of peripheral fibres is connected with the network, not with cells.

In the tubicolous polychaetes no neurogleia-network supports the central plexus, but it forms a coarse-meshed network for the support of the cells. In Lumbricus there is no neurogleia-network for either the cells or the nerve-plexus.

The colossal fibres of Lumbricus are surrounded by a delicate continuation of the central nerve-network; into this sheath they give short processes which divide dichotomously. As in other forms, therefore, they are constructed from the central nerve-network. The peripheral fibres arise, both directly from nerve-cells, and indirectly from nerve-plexus. There are no unipolar cells like those of polychaetes. The conspicuous median cells serve to connect together the right and left cell-regions.

The ganglia in Sipunculus are surrounded by two membranous coats. The space between the outer and inner coats is occupied by a coarse neurogleia-network, the meshes of which contain large cells. To the inner surface of the inner coat hangs a gleia-network of smaller mesh supporting the small nerve-cells. The inner network extends but a short distance into the central nerve-network. Direct anastomosis of the cells is common.

77. Histology of the Spinal Cord of Triton. BURCKHARDT (*Arch. f. Mikr. Anat.*, 34, 1, p. 131).

78. Histology of the Projections of the Epithelial Cells of the Ependyma. MAGINI (*Atti del Accad. Med. di Roma*, XV., May 4, 1890, p. 123).

In fœtal brains of many mammals Magini confirms Golgi's observations on the spinal cord of the hen, that the ependyma cells give off fine-branched processes, which reach to the dura mater. He sees therein a similarity with the neuro-epithelial cells of sense-organs, and thinks that the ependyma cells of the ventricles of the brain must represent an intra-cerebral sense organ. In the embryo the processes are varicose; with gold chloride and Golgi's method they stain like nerve fibres. They follow the course of the nerve fibres. They are more numerous in adult than in fœtal brains (of Man).

79. Histology of the Large Cells in the Anterior Horns. KRONTHAL (*Neurol. Centralbl.* 2, pp. 40-42, Jan. 15, 1890).

Method:—A piece of tissue the size of a small pin's head is taken from the fresh cord, placed on a slide, and squeezed into a thin layer by the cover-slip; 5 per cent. of watery solution of methyl-blue is placed at the edge of the cover-slip, which is then lifted so as to allow it to cover the film; the superfluous colour is drawn away by blotting paper, the cover-slip removed, and the film dried in the air and preserved in balsam.

Kronthal recognises the fibrillar structure of the processes and sees numerous fibrils which cross one another inside the cell. In some places a fibre could be followed which, entering a cell by a process, traversed the cell and entered another fibre.

80. The Double Refraction of Nerve Fibres. BECHTEREW (*Wjestnik Psichiatrii i Nevro-patologii*, vi., 2, 1889).

81. The Investigation of Nerve-tissue in Polarised Light. DIOMEDOWA (*Trans. Soc. Naturalists in the Imp. Univ. Kazan*, xxii., No. 4, pp. 180, figs. 7s. Russian).

82. The Presence of Ranvier's Constrictions in the Spinal Cord of Vertebrates. PORTER (*Q. Journal of Micros. Sci.*, cxxi., pp. 91-99, pl. xii. *bis*, April, 1890).

The observations of Torneaux, and le Goff and of Schiefferdecker confirmed, *contra* Boll and Kölliker. Method: Nitrate of silver 1 per cent. solution, osmic acid 2 per cent. solution, equal parts. Pieces of the white columns from near the anterior fissure immersed in the mixture for two hours, washed in dilute caustic potash, and teased in glycerin. It is especially difficult to exhibit the nodes of Ranvier in axial fibres owing to absence of a sheath of Schwann to support the myelin-sheath.

83. The Spiral Fibre and Peri-cellular Network of the Sympathetic Ganglion-cells. ARNOLD (*Anat. Anzeiger*, No. 7, 1890).

Maintains, *contra* Ranvier and Feist, that the appearances described by him are not artifacts.

84. Are Ganglion-Cells Amœboid? RABL-RÜCKHARD (*Neurol. Centralbl.*, 7, April 1, 1890, pp. 199-200).

An attempt to explain the origin of such exchanges as are at the bottom of psychical action by supposing that the fine nerve-network which is the seat and tract of such exchange, instead of having a defined invariable structure, fluctuates during life according to the connections which it establishes; the protoplasmic processes of the higher nerve-cells, from which the network originates, undergo amœboid changes.

HISTOGENY.

85. The Appearance of Caryokinesis in Cells of the Central Nervous System in new-born and young Dogs and Rabbits. BUCHHOLTZ (*Neurol. Centralbl.*, 5, Mar. 1, 1890, pp. 140-142).

After some account of the results hitherto obtained, the author describes the situations in which he has observed active cell division. Mitotic figures were seen in cells of blood-vessel walls, supporting tissue, ependyma of ventricles, cortex and great ganglia, but never in fully-developed nerve cells; the cells in which they were seen presented always a non-developed structure.

Method: Chromic acid—alcohol—hæmatoxylin and safranin in anilin-water after Zwaardemaker's method (*Zeitsch. f. Mikrosk*, iv., p. 212).

86. Origin and Ramifications of the Nerve Fibres of the Embryonic Spinal Cord. RAMON Y CAJAL (*Anat. Anz.*, v., Ni 3 and 4).

Observations on chicks by Golgi's method. Discovery of collateral branches which arise by a triangular swelling from the fibres of the white columns, and sink into the grey matter, where they end dendritically. The collateral branches of the anterior columns are the largest. They branch amongst the cells of the anterior horn. A small group from the most mesial fibres of the anterior column enter the anterior commissure. The collateral branches of the posterior column are finer and more numerous.

They traverse the substance of Rolando and enter the posterior horn. The writer looks upon these fibres as nerve connections associating distant clumps of grey matter.

The writer gives a complicated account of the connections of the nerve cells. He divides them into (1) cells of the commissure, (2) cells of the columns, (3) cells of the roots. The axis-cylinder processes of these three kinds of cells are connected with white fibres. (4) Cells with branched axis-cylinder processes are found in the posterior horn. Neurogleia-cells are transformed elements of the ependyma.

Monstrosity: Microcephalus.

87. The Brain of Microcephali. Giacomini (*Turin*, 1890, pp. 331, plates 23, figs. in text 14).

The first chapter contains an exhaustive description of nineteen cases of microcephalus, of which seventeen were collected by the author. In the author's cases the brain weight varied from 171 to 968 grammes. The second chapter is devoted to a *résumé* of the literature of microcephalus. The conclusions of most general interest are that microcephalus is not limited to any part of the central nervous system, but affects the whole; that it is always neural, not osteal, in origin; it is due to arrest of development, and varies in anatomical features in different cases according to the stage at which arrest occurred. The brain is not atavic, but belongs to the human type, and cannot, therefore, be used for determining the line of human descent.

Method.

88. Rapid Hardening of the Spinal Cord by means of an Electric Current. Minor (*Neurol. Centralbl.*, p. 294, May 15, 1890).

A piece of the spinal cord placed in bichromate of potassium solution, in contact with the positive pole of a circuit (*strength of battery not stated*), for four to five days is as well hardened as a similar piece left in the solution for two to three months without the current.

89. Section-series, with Photoxylin or Celloidin. Obregia (*Neurol. Centralbl.*, pp. 295-298, May 15, 1890).

Differs from Weigert's method chiefly in the use of a solution of sugar and dextrin (in water and alcohol) with which the object glass is covered. The sections are placed upon this glaze, and then covered with a solution of celloidin or photoxylin. They lie

therefore on a photoxylin membrane, not between two folds of celloidin. The riband is thinner, and the sections more easily stained.

90. The Staining of Nerve Tissue by Methyl-blue *intra vitam*. FEIST (*Arch. f. Anat. u. Phys., Anat. Abth.*, 1890).

After staining during life, the tissue is placed for some time in Hoyer's picrocarmine. A fibrillar structure of the centre of the axis-cylinder is shown in this way. By other methods the fibrils are shown as scattered throughout the whole cross section of the axis-cylinder. Transitional conditions were also observed.

A difference in reaction towards methyl-blue marks the olfactory fibres and other non-medullated fibres. Ehrlich's network surrounding the cells of the frog's sympathetic was seen as closed, and was in many cases directly continuous with the spinal fibre. Feist does not believe in the nervous nature of this network, but regards it as a reticular protoplasmic layer. In osmic-acid-maceration preparations he proved that the "straight" process branches.

91. Contributions to Histological Technology. MAYER (*Zeitschr. f. Mikroskopie*, vi., 4, 1890).

The action of methyl-blue when injected into the animal is not, as Ehrlich supposed, limited to life, for twenty-four hours after death it is effective. Perhaps this indicates that the nerves survive after general death. Positive and negative pictures are obtained, as with silver staining. Specimens can be preserved in a picric-glycerin mixture.

92. The Technik of Golgi's Staining. SEHRWALD (*Zeitschr. f. Mikroskopie*, vi.).

DANA ON THE PATH. AN. OF T.-D.

(*Journal of Nervous and Mental Disease*, Dec. 1890).

In a paper read before the New York Neurological Society, Dr. C. L. Dana expressed his belief that neuritis and degenerative changes in the nerve were not the usual and ordinary conditions in tic-douloureux, for in almost all of the cases, no matter how old, no permanent anæsthesia occurred. On the other hand, in cases of progressive trigeminal anæsthesia, due to degenerative neuritis, there had been but little pain. The question arose, therefore, whether there was any tangible cause of the condition. The author's proposition was that many cases were

due to an obliterating arteritis of the nutrient vessels of the nerve. His reasons for these views were:—1. That the disease occurred only at a time of life when degenerative changes in the arteries began. 2. That it affected chiefly and primarily one of the terminal branches of the internal maxillary. If it extended or recurred, it involved the inferior dental. It rarely affected seriously the supra-orbital nerve, which was supplied by a branch of the internal carotid. Hence the disease followed a certain fixed vascular distribution. 3. That he had examined four superior maxillary nerves, removed in typical cases of tic-douloureux; in none were there any noteworthy changes in the nerves. In three of them striking evidence of arterial disease was found. In the fourth case no blood-vessel was present in the specimen. 4. The view that an obliterating arteritis was a factor in this disease was strengthened by therapeutical experience. Nitro-glycerine would sometimes relieve pain instantly and prevent a return for a long period of time. Aconite, which was so useful in this disease, also lowered blood-tension; while potassium iodide, which sometimes favourably modified arterial disease, was occasionally useful in tic. 5. That there was unquestionable evidence that removal of the peripheral nerves sometimes cured tic entirely, and hence the disease was peripheral and due to some local peripheral irritation. 6. Certain authors had recently stated that by a new method of injection they had been able to discover a closer and more extensive relationship between the nerve trunks and blood-vessels than had hitherto been known, and they suggested in their conclusions that disturbances in blood supply might be a serious factor in causing neuralgia.

The author then gave the histories of a number of cases which he considered typically corroborative of his theory. He adduced positive facts that the trigeminus and its roots, and even nuclei and deep roots, were not diseased even in old and typical cases. In all cases where the vessels were examined, striking disease was found to be present. Circumstantial evidence was found by therapeutic experiment and the general etiology and anatomical distribution of the vessels and of the pains. His argument, he said, was defective, in that he had not examined the infra-orbital arteries of healthy persons of from forty to sixty years of age. But this was not much of a defect, for the reason that with obliterating arteritis there must be some specific vulnerability of the nerve centres. At present it seemed to the author that it would be pretty safe for him to assert that most of the typical cases of tic-douloureux occurring after middle life were due to an obliterating arteritis of the infra-orbital or inferior dental artery, terminal branches of the external carotid, plus some peculiar vulnerability of the central nervous system. Dr. Dana added that in such typical cases of chronic neuralgias—as, for instance, sciatica—evidences of arterial changes would be found, if sought for.